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CONTENTS

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BY JOHN C. THOROWGOOD, M.D., F.R.C.P.

CONVULSIVE SEIZURES
BY J. HUGHLINGS JACKSON, M.D., F.R.C.P.

SURGICAL TREATMENT OF DISEASES OF THE BRAIN
BY ERNST VON BERGMANN, BERLIN

INDEX FOR VOL. VI.

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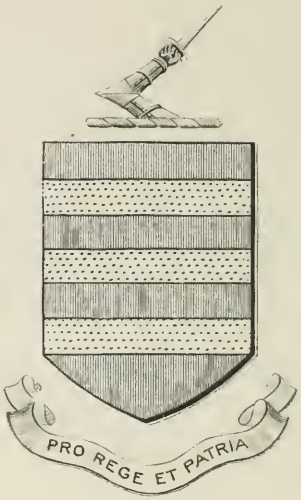

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WOOD'S

Medical and Surgical

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BRONCHIAL ASTHMA:[1]

ITS CAUSES, PATHOLOGY, AND TREATMENT.

CHAPTER I.

ON THE NATURE AND MODE OF PRODUCTION OF ASTHMA.

On receiving at your hands the appointment of Lettsomian Lecturer for the present year, my first thought naturally was as to the subject on which I might venture to address you with the prospect of being able to afford such information as would be of interest in its immediate delivery, and of practical utility hereafter.

Twenty-one years' experience among patients at a hospital specially devoted to all forms of chest disease, ought, I considered, to supply material from which to make three chapters: and I have accordingly taken as my subject a disorder of the breathing apparatus, which has long interested me much, and of which, as illustrated in a large number of cases, I possess numerous records. In choosing Bronchial Asthma as my subject, I purpose to speak, first, of the history, nature, and mode of production of this disorder, endeavoring thus to establish a clear definition of the malady; and then to discuss, and, as far as I can, explain, the various and conflicting theories that have been propounded as to the seat and nature of the asthmatic spasm. Secondly, I shall have something to say regarding some special varieties of asthma, and then shall pass to the consideration of bronchial asthma as a secondary neurosis, with its pathological relations and affinities. The third and last chapter will be on the treatment of asthma for purposes of prevention, palliation, and cure.

bladder, the bellows would be opened but a little way, and would inspire difficultly. So it appears in the business of the asthma, the inspiration is difficult and laborious, because but little air can be admitted into the contracted bronchia, and the vesiculæ drawn up. This puts the scapular and intercostal muscles and diaphragm upon a violent endeavor to press in the air and open the lungs, which nisus authors have mistaken, and supposed the pneumonic muscles, especially the diaphragm, to be convulsively affected; but it may easily be apprehended that the diaphragm cannot press the viscera downward to enlarge the breast if the air cannot be admitted into the lungs to follow its depression and fill the cavity of the breast; and this is the true reason why the diaphragm cannot move in the asthmatic fit. The contraction and stiffness of the lungs during asthma causes a catalepsis or rigidity of the diaphragm—the part most unjustly accused of this tyrannic oppression."

Thus we see that Floyer, while fully cognizant of the rigid state of the diaphragm during a fit of asthma, does not agree with those of the present day who, like Wintrich, Bamberger, and Lehmann, believe a tonic spasm of the diaphragm to be the essential cause of asthma. Floyer considered the wheezing noise in expiration during asthma to be due to narrowing of the smaller air-tubes in consequence of contraction of their muscular fibres. That this symptom is not due to phlegm in the tubes, he says, is evident, because the hysteric, who have no phlegm, wheeze much.

Floyer was in error in assuming the presence of muscular fibre in the wall of the pulmonary vesicle. The small bronchi are muscular and elastic—the cells are elastic, but not muscular. What Floyer meant by inflation of the membranes around the lungs by humors, I take to mean bronchial œdema and congestion; but that he regarded the contraction as brought about by mechanical pressure rather than by nervous influence seems clear, from his saying that certain writers of his day, Van Helmont and Dr. Willis, were wrong in regarding asthma as a convulsion, to be treated by anti-spasmodics, hot tinctures, gums, volatile salts, and sulphur medicines. The proper method is by evacuants, and remedies calculated to promote secretion and effect the discharge of humors—anti-congestive rather than anti-spasmodic treatment.

Passing on from Floyer to Dr. William Cullen, the distinguished nosologist of Edinburgh, we find him, in his "Synopsis Nosologiæ Methodicæ," published in 1772, defining asthma as "spirandi difficultas, cum angustiæ in pectore sensu, per intervalla subiens." Cullen appears to have regarded spasmodic asthma as essentially due to constriction of the bronchial muscles, and in his nosology he divides asthma into three groups.

First—Idiopathic asthma, of which kind he enumerates eight varieties.

Second—Symptomatic asthma, two varieties, viz.: 1. Gouty, asthma arthriticum; 2. Syphilitic, asthma venereum.

Third—comes a long list of asthmas, dyspnœas, and orthopnœas symptomatic of cardiac and pulmonary difficulties and obstructions.

The belief held by Floyer and Cullen that the spasm of bronchial asthma was due to contraction of the smaller bronchial tubes receives plenty of support in the present day. Our late distinguished Fellow, Dr. Hyde Salter, has expressed himself strongly and decidedly as an upholder of the bronchial constriction theory, and among other celebrated men of the same way of thinking I may name Trousseau, Romberg, Bergson, Biermer, Théry, Williams, Walshe, Fuller, Watson, Radclyffe Hall, Gerlach, MacGillivray, and others.

Sir Thomas Watson describes asthma as a difficulty of breathing occurring in paroxysms, and accompanied by loud wheezing sounds "in the respiration; going off after some hours, with more or less mucous expectoration, and unattended by fever. And these paroxysms of dyspnœa are believed to depend upon a spasmodic constriction of the bronchial tubes."

There comes next to be noticed, in the history of the growth of our knowledge of asthma, the theory put forward and ingeniously supported by Dr. Robert Bree in 1801. Dr. Bree, when in considerable practice as a physician at Leicester, was attacked so severely by paroxysms of asthma, that in 1793 he felt compelled to abandon his profession and accept a commission as captain in a regiment of militia. Under these altered conditions of life Dr. Bree lost his asthma—to return, however, some years later with great severity, rendering the latter portion of his life a period of constant suffering, and at

times of the most agonizing distress. In 1796 Dr. Bree resumed the practice of his profession in Birmingham, and while there he published his treatise entitled "A Practical Inquiry into Disordered Respiration, distinguishing the species of Convulsive Asthma, their causes and indications of cure." This work reached a fifth edition, and conferred upon its author a very high reputation, while it brought him also considerable practice. In 1833 Dr. Bree died at his house in Park Square, Regent's Park, at the advanced age of eighty years. The great point in Dr. Bree's theory of the cause of the asthmatic paroxysm was the presence in the air-tubes of some peccant irritating matter which excited the lungs powerfully to effect its discharge. When, by the expulsive efforts of expectoration, the peccant mucus was thrown off, then the spasm in the air-tubes came to an end, the desired object having been attained in the expulsion of the mucus. Dr. Bree had observed carefully the facts of an asthmatic paroxysm, but had not correctly interpreted these facts; for, as Salter has well shown, during the asthmatic fit the power of discharge from the lungs by cough is quite in abeyance. Moreover, I hope, as I proceed, to show that the collected mucus is the effect, and not the cause, of the asthmatic spasm.

He who, in the present day, is the nearest exponent of Dr. Bree's theory is Leyden. This observer found, in the viscid gray mucus expectorated on the subsidence of a fit of asthma, brownish masses of cells undergoing granular degeneration, and among these could be made out colorless octahedral crystals soluble in hot water and in acids and alkalies, but not in ether, and consisting probably of a substance analogous to mucin. Leyden believes that these sharp crystals irritate the peripheral extremities of the vagus nerve, and produce reflex spasm of the bronchial muscle.

Attempts to prove the connection by experiment appear to have failed, and the presence of these angular crystals is not peculiar to the sputum of asthma, for similar crystals have been found in the expectoration of persons suffering with ordinary catarrh and croupous bronchitis. Leyden possessed the knowledge of reflex nervous spasm, unknown in the time when Dr. Bree wrote; and I need hardly say that it is to the discovery of excito-motory and reflex nervous action, as well as of auscultation by the stethoscope, that we now owe so

much of our knowledge of the true nature and definite varieties of asthma.

Persistent mechanical irritation of the air-tubes by the inhalation of various kinds of dust will produce irritability, swelling, and various degrees of thickening in the lining membrane of these tubes; but asthmatic paroxysms are by no means a constant accompaniment of these pathological conditions.

Workers at dusty employments, millstone-dressers, knife-grinders, and chaff-cutters, get their lungs greatly irritated by the dust and fine steel particles they inhale while at work, and the effect is very obstinate chronic bronchitis. Asthmatic paroxysms may co-exist, but such co-existence is certainly not an invariable rule, and depends on the presence or absence of a neurotic or spasmodic element in the case. In six cases of mechanical bronchitis, detailed by Dr. Headlam Greenhow, I find no mention of distinct asthmatic attacks. The tendency of the disease was toward change of structure, rather than to nervous spasm. The trustworthy authority just quoted, in speaking of the asthmatic complications of the so-called "dry catarrh" of Laennec, attributes the dyspnœa partly to narrowing of the bronchi by swelling of their lining mucous membrane, and partly also to spasm of these tubes. That irritation leading to local congestion of the pulmonary mucous surfaces may become the cause of an acute attack of dyspnœa I do not doubt; but before entering further upon this question, I wish to place before you the reasons that make me uphold the doctrine of the existence of an essential independent spasmodic bronchial asthma, dynamic in nature and symptomatic only of some great nervous perturbation in the system; at times centric and emotional, at other times reflex, in its mode of origin; capable, too, of artificial production by direct irritation applied to the trunk of the vagus nerve.

The reasons that have prevailed to establish and maintain in my mind the conviction that we can have a severe spasmodic dyspnœa of purely nervous origin may be divided into two groups:

First—The evidence adduced from observation of cases as regards causation and cure.

Second—The support given by experimental physiology to the belief in nervous spasm as the cause of bronchial constriction.

My own observation has shown me that a most severe fit of bronchial asthma, requiring the inhalation of chloroform for its relief, may develop in the space of two minutes. In another case, a youth going up for examination at Burlington House is breathing with comfort, when the anxiety due to the non-arrival of the carriage to convey him to the place of examination causes a speedy attack of asthma, which subsides as the carriage draws up at the door.

Dr. Chowne reported a case where a dash of cold water over the foot invariably and immediately induced a paroxysm of asthma in a gentleman fifty years old. Dr. Salter affirms that he has seen severe asthma developed in as short a time as thirty seconds.

In 1834 John Ross reported a sudden and severe paroxysm of asthma, brought on by the smell of a parcel of new feathers.

The late distinguished French physician, Trousseau, who was the subject of inherited asthma, used to relate how the worst attack of asthma he ever experienced came on in the course of a few minutes, with intense dyspnœa and oppression, relieved in eight or ten minutes by smoking a cigar. Trousseau was at the time of his seizure up in the hay-loft watching his coachman, whom he suspected of dishonesty, measuring oats, and he attributed his severe attack to emotional agitation. "It could not have been the dust of the oats," says Trousseau, "for I have often been exposed to far more dust, and yet had no asthma." The profuse running from the eyes and nose, however, that Trousseau had, make me think his seizure was a sort of catarrhal asthma, due to inhaling dry pollen in the oat dust; this might have caused the profuse coryza; the asthma, doubtless, was emotional and spasmodic.

Trousseau gives a good example of the nervous nature of asthma in the case of the brother of the Chancellor of the Chamber of Peers in Paris, who used to free himself at once from his difficult breathing by having four or five bright lights brought into his apartment. A bad asthmatic, whom I have known for years, declares to me that the contemplation of himself in a looking-glass constantly gives relief to some of his worst attacks. With some the mere presence of a cat, a dog, or a hare-skin, in a room, at once brings on asthma.

Walshe gives a striking instance of asthma occurring as a

primary neurosis of emotional, or centric, origin in the case of a man, very liable to spasmodic asthma, who always carried in his pocket some cigarettes made of belladonna and stramonium. One day, at a distance from home, he suddenly found he had forgotten his cigarettes. Instantly he had a violent paroxysm of asthma.

Salter has noticed asthmatic seizures to alternate with those of epilepsy; and Eulenberg has seen paroxysmal breath seizures occur in alternation with hemicrania and angina pectoris.

The great relief to the asthmatic fit, often obtained by the inhalation of the fumes of tobacco, stramonium, and belladonna, and the speedy way in which chloroform vapor dissipates the spasm, further support the belief in the neurotic character of the seizure; and the more the nervous spasm predominates, the more certain is the relief obtained by the use of the above-named agents—a point I hope to work out satisfactorily when I come to the matter of therapeutics in my third chapter.

Observations that I have had opportunities of recording have shown that there is no rise of body temperature during a night of intensely severe asthma, brought on by nervous excitement. Such are a few of the facts that make me think we must admit into our nosology bronchial asthma as an essential primary neurosis. Now comes group of evidence No. 2, based on physiological experiment, as well as on clinical observation; and here I approach the question, so warmly contested just now, as to what part of the respiratory mechanism is the seat of the nervous spasm. Let us look first at the result of experiment. We find in the very smallest bronchial tubes a distinct layer of smooth muscular fibres disposed between an external and internal layer of elastic fibres. These elastic fibres support the borders and angles where several alveoli meet, and they are further distributed in a retiform manner over the alveoli of the lung. This membranous wall of the air-cell, according to Stricker, is entirely devoid of all trace of muscular fibre, and the air-cells were found by Dr. Williams, in 1841, to possess little contractile force under the influence of galvanic or chemical excitation, though the lung by its elasticity is capable of contracting generally to about one-third of its ordinary bulk when the thorax is opened.

paroxysm, as I hope shortly to show you. To me it appears that both respiratory acts are impeded more or less. Sometimes inspiratory difficulty is most urgent; at other times, and in other cases, expiratory effort is pronounced and unmistakable. My own reports of cases show me one group of patients who unhesitatingly refer their breath difficulty to the act of inspiration; others, among whom I am sorry to have to place an eminent member of our profession, say that their sufferings during the paroxysm of asthma are so intense that they hardly can tell whether inspiration or expiration is most laborious. A third group, to be treated of more fully in another chapter, often spontaneously affirm that the desire they have is to get the air out of the chest rather than to get it in.

In proportion as lung emphysema is developed, especially if slight bronchitis co-exist, so does the expiratory dyspnœa obtain the ascendancy. In young persons, where spasm is marked without much emphysema, inspiratory dyspnœa is often most severe and distressing; and not long since I saw in the case of a middle-aged man, who had long been afflicted with most severe attacks of almost asphyxiating asthma, a good example of inspiratory dyspnœa. "Decidedly," said he, "my difficulty is in inspiring."

In this case, I observed thoracic distention was in no way marked, and on listening to his chest six hours after one of his very bad attacks, I could hear no sibilant expiratory wheezings, and expiration seemed free. I further noticed that the percussion resonance did not come up to the normal standard of clearness, and this imperfect resonance of chest after a fit was remarked on by the patient himself. The patient was a strong, vigorous man, with no sign of degeneration of tissue, and his asthma resembled that which one observes in quite young persons. I have been struck with the fact, just exemplified, that on examining the chests of patients of the class described, where inspiratory distress is intense, and chest distention by no means marked, I have failed on the day following a bad night of asthma, to find about the chest any of that prolonged sonoro-sibilant expiration with which we are so familiar when we listen to the chest of an asthmatic shortly after his fit. Another point I have investigated, is the position of the diaphragm in these cases, and have found it to be

drawn up rather than pressed down. The shoulders of the patient are nearly always high, for the thorax is drawn powerfully up by the muscles of inspiration. The normal movement of the ribs outward and forward does not take place, so that true divergence of ribs and expansion movement is not appreciated by the applied fingers. I purpose soon, after I have read the notes of a case, to return more fully to these points; meantime, I would observe how a great inspiratory effort can be made by drawing up of the thorax in the way just described; and also how a similar effort can be made, of a more abdominal character, in the performance of which the diaphragm descends and the abdomen protrudes; but in this last effort the lung must be fully expanded, and if dyspnœa occur it will be more of an expiratory than of an inspiratory character.

I certainly think that either of these conditions of thorax and abdomen may obtain in cases of asthma. In each case the muscles of inspiration act to aid the elastic resilience of the chest-wall, which Dr. Douglas Powell has demonstrated as an active force in ordinary tranquil inspiration. The embarrassed breathing and asthma are due to want of corresponding action between the lungs and the thorax, for these organs have their expansive power limited by the spasm of the bronchial muscle and cannot follow the chest-walls as they expand. One is reminded of Floyer's simile of the bladder tied within the bellows (see p. 647). The lungs are in arrest of action, and in the first case, where chest and diaphragm are drawn up, the air is in a great measure evacuated from the lower part of the lungs, and is kept out by the bronchial stricture; hence the straining inspiratory effort, while the pulmonary spasm causes a sense of tightness or tearing in the upper chest, and here, indeed, air may be held pressed into the apices of the lungs, just as it is pressed into these parts by the violent expiratory efforts of coughing. In the other case, where the diaphragm is down and chest overfull with air, we get very prolonged expiratory wheezings, the bronchial spasm retarding expiration. As I have said, I shall hope shortly to follow these views a little further; being desirous now of placing them as simply as I can, I make no reference to imperfect aëration of blood in the lungs as a possible cause of determining, in a reflex way, powerful inspira-

tory efforts; and, speaking of inspiratory difficulty, I of course exclude all laryngeal or tracheal disease in the way of congestion or chronic stenosis of these parts. I have endeavored, by an analysis of the notes of a number of cases, to illustrate the varying conditions of the chest and abdomen during the asthmatic fit, and thus to explain how it is that authors differ so decidedly in the descriptions they give of the state of the thorax during such fit.

This same impression of want of unanimity of description seems to have been made on the mind of Dr. Berkart, the most recent writer on asthma, and he has placed the various descriptions so well together that I cannot do better than quote them just as they are given, with the authorities.

Respiration retarded to from 9 to 7 per minute.—Salter 77.

The lungs are unusually collapsed within the thoracic cavity, and the parietes drawn in.—Williams, 91.

Inspiration is difficult; expiration, short, easy, superficial.—Bergson.

Thorax sounds dull on percussion.—Williams.

Respiration augmented to 40, 60 or 80 per minute.—Canstatt, Ferrus, Wunderlich, and J. C. Thorowgood.

The chest remains distended, its walls are kept fixed in the extreme inspiratory position, such enlargement involving all the diameters of the thorax.—Salter, 76.

Expiration mainly is impeded, the patient labors 4 to 5 seconds to empty his chest.—Biermer.

Percussion induces a hyper-resonant note.—Biermer.

While inspiration shows nothing that varies from an ordinary powerful and somewhat forced effort, expiration presents the picture of a most laborious and tormenting, and at the same time fruitless, struggle. (Bamberger: "Ziemssen," IV., 562.)

Inspiration is violent and painfully ineffective; expiration is much prolonged, but unaccompanied with notable anguish (Walshe).

Some observers, I find, speak of the abdomen as distended, others as retracted; the fact being, as I have shown, that sometimes one condition, sometimes the other obtains.

I remember, some years ago, when first my interest in the nature of the asthmatic paroxysm was aroused, thinking that sufficient attention had not been paid to the part taken by the diaphragm in the mechanism of asthma. That this muscle is much concerned in the dyspnœa of old emphysematous

persons, I fully believe; but I cannot agree with Wintrich and Bamberger, who deny the influence of bronchial stricture in producing asthma, and attribute the seizure to tonic spasm of the diaphragm. True diaphragmatic spasm is seen best in some forms of hysteria, and it is marked by short, hurried inspiration, short pause, and rapid expiration. In a case of this description, shown to me by my clinical assistant, Mr. MacDonald, the respirations were as many as eighty in the minute; a short, quick movement at the epigastrium was observed; auscultation revealed no abnormal sound. The patient was a young woman, and had had similar attacks before during the last few years. Bamberger believes, with Wintrich, that with a depressed position of the diaphragm there can be no bronchial stricture. Wintrich, moreover, says it is impossible to explain the enlargement of the lungs with increased air contents by a spasmodic contraction of the bronchial muscles. Bamberger further says that bronchial spasm is in the highest degree improbable, if not unimaginable, for in such case, inspiration and expiration, especially the former, must be laborious and prolonged. Against these statements we have the fact, testified by several highly competent to judge, that inspiration and expiration in asthma are both very laborious acts; and we find Biermer stating, most correctly as I think, that a depressed condition of the diaphragm and acute pulmonary distention are quite compatible with bronchial spasm. I have myself during the last few years seen many cases of most severe asthma, with every evidence of bronchial spasm, where the chest was large and round, and the diaphragm undoubtedly depressed. During the intervals of actual spasm very little air movement can be heard on listening to the chest of such patients, and this is an unfavorable sign.

Acute pulmonary dilatation follows on acute obstruction, just as chronic pulmonary dilatation and emphysema follow on obstruction of air-tubes by old bronchial catarrh (Ziemssen, 543).

Before I had an opportunity of studying the observations of the writers just quoted, I had given up my idea of diaphragmatic spasm as a cause of asthma, while I came to think that a kind of inertia, or partial paralysis, of the diaphragm may have a good deal to do with the expiratory dyspnœa of old emphysematous persons.

From what I have observed in cases of spasmodic asthma coming on very suddenly, and sometimes brought on by some great expiratory effort, such as a fit of violent coughing or laughing, it seems that the thorax is drawn up and fixed; but on looking at the lower part of the thorax, quick, short, and very limited movement of the diaphragm and lower ribs may be observed, by which respiration is kept going and life maintained. The distress in cases of this kind, often met with in young persons, is markedly spasmodic and most severe, so that chloroform inhalation is often the only agent that gives effectual relief.

Allow me here to introduce my record of a typical case of this form of asthma:

A young man in good health had his first attack of asthma take him quite suddenly after a fit of excessive laughter. Here was an example of spasmodic arrest of breathing after a complete expiration. On another occasion I saw this youth pass, in the course of two minutes, from sudden exposure to cold to the surface, into an intense and even alarming paroxysm of asthma, and during this seizure I noticed the state of the chest. There was great drawing up of clavicles and thorax, with fixation of these parts. The abdomen, at xiphoid cartilage, was powerfuly retracted, and the diaphragm elevated. Respiration counted 64 per minute; pulse 110, very small. The breathing act seemed carried on by a slight, rapid, and very limited movement of the diaphragm and lower ribs. There was no laryngeal difficulty; chest resonance, deficient in clearness; expiration, wheezy, jerky, and prolonged; inspiration, short, and quickly followed by expiration. The inhalation of chloroform soon gave relief to the spasm, and the patient was released from his fixed bent position over a table, which he assumed on the fit taking him.

This young man found, like many other cases that I have observed, that when he was able to cough and get up some mucus he felt better; and a pill of pil. hydrargyri and pulvis scillæ gave notable relief, apparently by promoting expectoration. Dr. Bree would explain this by saying, "Your pill fetches up the irritating mucus from the lungs, and so does good to the asthma;" others would very reasonably attribute the benefit derived from the pill to its relieving congestion of the bronchial mucous membrane. Against the exclusive

truth of the theory of congested mucous membrane as the sole cause of dyspnœa, I would urge that the congestion and subsequent mucous flux result from the pressure put upon the capillary vessels by the spasm of the bronchial muscles. The great suddenness of the invasion of asthmatic dyspnœa, compelling the patient to leap from his bed and rush to the open window, and the speedy relief often afforded by inhaling the vapor of ether or chloroform, or the smoke of burning tobacco or stramonium, point to nervous spasm rather than to acute congestion. The spasmodic constriction of the bronchial muscle induces stasis of circulation and congestion, and if flux and secretion can be determined, the patient feels much relief. Thus, I have often observed the spasm of asthma to subside when bronchitis comes on and causes free secretion; and people say, "When I get bronchitis, my asthma goes away." In the essential dynamic form of bronchial asthma, as illustrated in the case the notes of which I have just read, there is to all appearance, as well as on the emphatic declaration of the patient, marked inspiratory dyspnœa. Seeing how often these intense fits of bronchial spasm follow on an exhaustive expiratory effort, one can comprehend the inspiratory character of the breath difficulty. Previous expiration has pumped the air out of the lower part of the lungs, and forced some up into the apices, just as we see in the expiratory effort of coughing; here this air is held, for the thorax is raised and drawn up in powerful inspiratory effort, and the tension in the upper chest becomes very distressing; meantime, the retracted condition of the lower part of the lung causes the diaphragm to rise up with the thorax. Patients under the influence of this typical form of bronchial spasm have volunteered to me the statement that they feel as if their lungs were drawn up—drawn up to choke them, as Floyer used to say. A young man described his fit to me also in these words: "Cold air seems to draw together and contract my air-tubes, and then I pass into one of my bad fits;" and the fits in this case were as intense as any I have met with, setting at defiance the best-planned remedies of numerous physicians. It is in the young we often see these very severe attacks of bronchial spasm. Dr. Fuller says that the most frightful paroxysm of asthma he ever saw was in a child æt. thirteen. Salter describes an alarming attack in an infant not two years old, and I expect

not uncommonly the bad pulmonary attacks of infants during dentition, if carefully investigated, would be found to have more of the element of spasm than of inflammation about them. The drawing up of the diaphragm was well exemplified in the instance of an asthmatic little boy who used to gasp out, "See how hollow I am!" as he pointed to his retracted epigastrium. With respect to this elevation of the diaphragm, I find that Wood, of Philadelphia, describing a fit of asthma, says: "A hollow in the epigastrium is produced by the unusual elevation of the diaphragm, while expiration is much less difficult than inspiration." Walshe, too, already quoted, says: "Efforts at inspiration, convulsively violent and painfully ineffective, are accompanied, in the purely dynamic form of the disease, with sinking of the epigastrium, falling in of the lower part of the sternum, and elevation of the diaphragm. Expiration in these cases is prolonged, but unaccompanied by notable anguish" (p. 546, 4th edition).

In these cases of asthma in young persons not due to whooping-cough or antecedent bronchitis, and very prone to come on after an expiratory effort, the prognosis should be favorable; for though inspiratory spasm is alarming, with great sensation of internal constriction or cramp within the chest, yet the chance of co-existing emphysema is not great, and we may see in impairment of percussion resonance, elevation of diaphragm and absence of excessive chest distention, evidence on which to found a hopeful prognosis.

The fact—and of this I become more and more convinced—that young people do grow out of this uncomplicated spasmodic asthma, or, better still, perhaps, that they can be cured of it, if it be due to some reflex irritation—as worms in the bowels, or enlarged bronchial or tracheal glands in the chest—shows that no high degree of emphysema can have been produced. "The asthma of children," says Riegel, "often ceases spontaneously after a short time, or else at the period of puberty." (Ziemssen, IV., 566.)

The theory of Lebert that the spasm beginning in the bronchial muscles, and thus narrowing the tubes, causes a great excess of inspiratory effort to force the air through the constricted bronchi, and then that this increased respiratory stimulus, reflected to the medulla and respiratory centres, induces reflex spasm of the diaphragm through the agency of

the phrenic nerve, is ingenious and worthy of notice. The theory appears to accord with the fact that we see cases of what I call ingravescent or culminating asthma, slight at first, but getting gradually worse: want of air increases till an actual state bordering on true apnœa is produced; then come on most violent inspiratory efforts, and every auxiliary muscle is called into violent action. The power of these extreme inspiratory efforts was demonstrated by the experiments on asphyxia made before the Royal Medico-Chirurgical Society a few years ago. These facts show how wise it is to try and cut the asthmatic fit short *in initio* by anti-spasmodic inhalation in the early stage. Experienced asthmatics know this well. "*Obsta principiis*" is their motto.

The theory of Weber and Störck, already casually referred to, that asthma is due to tumefaction of the bronchial mucous membrane in consequence of dilatation of the blood-vessels by vaso-motor nervous influence, is full of interest and importance, especially in connection with certain varieties of asthma and catarrh, to be treated of in Chapter II. Störck says he has seen the mucous membrane of the larynx, trachea, and right bronchus in a state of hyperæmia during an attack of asthma. To see as much as this in the windpipe of an asthmatic during his fit reflects credit on the patience and perseverance of observer and observed, and I am inclined to think that any one who can manage to keep the laryngeal mirror in the throat of an asthmatic for but a short time will be pretty sure to obtain evidence of congestive hyperæmia. Be this as it may, there is the oft-made complaint of asthmatics of tightness and obstruction in the nose, the tendency often to profuse nasal flux, and the liability at times to the formation of mucous polypi in the nasal passages, all pointing to a congestive hyperæmia of these parts.

My reasons for believing the congestive hyperæmia and flux to be secondary effects of nervous spasm have been already set forth. Biermer, whose views on the nature of asthma seem to be fully in accord with the results of experiments and the observation of clinical facts, and who, like myself, is a supporter of the bronchial-spasm theory, regards a fluxionary hyperæmia of the bronchial mucous membrane as quite a possible cause of asthma.

I agree in this, as in most other points, with the excellent

observer just named; and, in considering the therapeutics of asthma, it is well to be mindful of the condition of bronchial hyperæmia, be it primary or secondary. Furthermore, for purposes of treatment, it is of the greatest importance to discover whether the breath difficulty be one of inspiration or of expiration.

CHAPTER II.

BRONCHIAL CONGESTION, AND THE PATHOLOGICAL RELATIONS OF ASTHMA.

In my last chapter I placed before you the most prominent theories that have been advanced in explanation of the phenomena attendant on a fit of spasmodic bronchial asthma. I sought to make clear, from experimental and clinical evidence, the reasons that support my belief in the neurotic or spasmodic nature of the breath stoppage; and then I discussed the question of the seat of this spasm, showing that most of those who, for the last century, have given their attention to asthma, believe the spasm mainly to affect the smooth muscular coat of the bronchial tubes. The part taken by the diaphragm in the mechanism of asthma was also considered, and I endeavored to prove that the condition of this important muscular structure during a fit of asthma, with respect to elevation or depression, is not a constant one; but sometimes it may be elevated, sometimes depressed, according to the nature and rhythm of the respiratory effort. The cases where the diaphragm is raised, and the epigastrium retracted, appeared to be most common in young persons, and in hysterical subjects, where inspiratory dyspnœa was marked, where no great degree of emphysema was developed, and where the asthma was often excited by some powerful expiratory effort, as, for example, a great fit of coughing or laughing. The cases where the abdomen protruded, and the diaphragm was depressed, were usually those of persons who had more or less emphysema of lung, whose chests were overfilled with air, and whose respiratory difficulty was rather in expiration than in inspiration. In the recognition of these features, I looked to find some explanation of the contradictory statements of many authorities as to the condition of the thorax and lungs during a fit of asthma. How far I have succeeded in establishing the truth of what I have advanced, I will not

stay to inquire; but I think we must all agree that the subject is one on which, in our present state of knowledge, some difference of opinion must be allowed, and we must not expect the term "asthma" to comprehend a complete uniform train of symptoms. No wonder, when we reflect on the complex nature of the respiratory process, and the various forces and conditions involved in its performance.

We now pass from the nervous and muscular elements of the asthmatic fit to enter more fully on the vascular element —on congestion of the air-tubes as a producer of severe dyspnœa. I have already put before you the theory, propounded by Weber, in the form of a supposition of tumefaction of the mucous membrane of the bronchi in consequence of dilatation of the blood-vessels through vaso-motor nervous influence. This idea of rapid congestion causing serious dyspnœa does not lack support. There are those who appear to regard it as the exclusive cause of asthma—at least, so I judge from some of the statements I find put forth by writers. Professor Sée, of Paris, for example, in a very interesting paper in the "Practitioner," on the "Use of Belladonna in Asthma," concludes with these words: "We need not concern ourselves with the bronchial muscles, as it is well understood at the present day (1869) that they play no part in the production of asthma." The last time I ever saw Dr. Hyde Salter smile was when I read to him these remarkable words of the French professor; but Salter said he was aware a similar opinion had been expressed by others in a way by no means complimentary to the upholders of the doctrine of bronchial spasm.

Weber, who appears the original propounder of this bronchial-congestion theory, is far from denying bronchial spasm as a factor in the asthmatic fit; and Biermer, whose rational and clear views it is a pleasure and satisfaction to study, attributes a certain *rôle* to the fluxionary element in bronchial asthma, while he also supports the doctrine of tonic bronchial spasm.

In old days we know that a distinction was drawn between dry and humoral asthma; and nowadays we seek to make this differentiation more evident, and to place it on a sound basis. Purely nervous asthma of reflex origin, as, for example, hysterical asthma (and you may remember how I quoted from Floyer's old book the words, "that the hysteric who wheeze

much have no phlegm"), I believe to be independent of congestion and due purely to spasm; while, on the other hand, there must be admitted to exist a class of cases where congestion, hyperæmia, and mucous flux are undoubtedly found playing a very important part in causing dyspnœa, and guiding us, moreover, materially as to our method of treatment.

I may just instance the congestive bronchitis and dyspnœa of cardiac disease. The gasping and orthopnœa of the state that we call cardiac asthma, may be due entirely to bronchial congestion; and I have seen, as others also must have seen, cases of this description of congestive asthma who have been thoughtlessly told to smoke tobacco and stramonium, and who come and tell us that the smoking makes them worse, and to feel as if they would die in a faint. We may get at times attacks of muscular spasm in these cases, caused by the vascular congestion disturbing the rhythmical action of the bronchial muscle, and only then is it that anti-spasmodics may be temporarily needed. Salter was very strong on the sympathy of the bronchial muscle with the irritated mucous membrane, and went even so far as to express his "belief that severe chronic bronchitis never exists without asthmatic complication;" a belief in which I am far from agreeing with my late valued friend.

I pass, for a time, from the consideration of bronchial spasm caused by the inflamed, congested, and irritated condition of the superjacent mucous membrane, to speak of some varieties of asthma and bronchial catarrh, due to certain exciting substances applied to the mucous membrane itself. Specific irritants acting on an unduly sensitive mucous membrane may first of all bring about rapid congestion with profuse catarrh and flux, and then, after a period of varying duration, bronchial spasm and asthma come in as secondary effects. I will briefly describe the symptoms and progress of an attack of summer catarrh, or hay asthma, as in this affection we have an admirable example of the points to which I wish to draw attention.

A small boy, who had enjoyed up to the age of eight years very good health, was playing in a hay-field in the month of June, when suddenly he was seized with profuse flow of tears, and swelling of the eyelids, to an extent of well-nigh blinding him; at the same time came on a violent fit of sneezing. He

was taken by his companions into the house, and soon recovered. These attacks continued to recur in the summer whenever the patient was exposed to their exciting cause; but what is of especial interest in the case is the fact that it was not till he was twenty years old that true asthma and breath difficulty were added to the already established catarrh. "Asthma at night," says the patient, "becomes my great touble toward the beginning of June; after a few whiffs of a cigar I can get my breath easier and manage a deep inspiration which seems to burst open the contracted bronchial tubes, but they soon contract again, and reset themselves at a diminished calibre. That which gives real and continued aid in my distress is smoking strong tobacco in a pipe until I am approaching a condition of collapse. A failing pulse and a damp, sweating skin are always accompanied by marked alleviation of the sufferings, the relaxation of the contractile tendency affecting the bronchial as well as the other muscles." (Salter, 328.)

Here we have, then, an excellent example of hyperæmia and flux, due to direct irritation of the mucous membrane, existing for some years, and eventually complicated with a very severe form of spasmodic bronchial asthma.

Other forms of congestive catarrh and spasm of similar type are seen in examples of asthma caused by the aroma of fresh coffee, by inhaling the dust of lamp-black, of ipecacuanha powder, and of linseed and mustard meal. Two cases of very severe spasmodic asthma, due to excitation of the naso-pulmonary mucous membrane by these two last-named agents, have come under my notice. One patient, recently under observation, begged me on no account to prescribe ipecacuanha in any form; "for once," said he, "I had fifteen drops of the wine of ipecacuanha given to me, and it brought on such a paroxysm of asthma, that I was well-nigh choked, my lower chest seemed powerfully retracted, the difficulty of drawing in my breath was extreme, and perspiration rolled off from me." Here the ipecacuanha wine might have acted in a reflex way, through the gastric branches of the vagus nerve.

When we examine into the history of cases of genuine hay asthma and summer catarrh, we find nearly always that the coryza and catarrh precede the asthma. Possibly, as in the case lately narrated, several years may elapse before the

summer catarrh becomes complicated with breath difficulty and spasm.

Another point, brought out by inquiry into the history of cases of hay asthma, is the ingravescent nature of the affection. It grows worse with every fresh attack, and the susceptibility to the exciting cause, instead of diminishing with exposure and lapse of time, increases and becomes more deeply rooted in the system. The blood-vessels more readily congest, and the congestion goes deeper down into the air-tubes. The sudden advent of the catarrh is familiar to all, and one need not go far to find those who can describe all the distress attendant on the attack. A lively and telling account of catarrhus æstivus, hay, rose, or peach, cold or asthma, is given by Dr. Dobell, in his work on "Winter Cough," and is a lively bit of reading, in full detail. "The cross of my life," says the writer, quoted by Dobell, "is DUST, and I print the word in capitals. Once exposed to dust, every particle is to my eyes as a grain of cayenne pepper; and itching eyes, snuffing, sneezing, and vexed temper, are at once my lot." The writer then goes on to describe the dyspnœa which follows on the catarrhal state, and presents the usual characters of bronchial asthma.

The catarrh begins in the eyes and nasal passages, and we may get speedy asthmatic complication of reflex type, owing to irritation of the Schneiderian membrane. Then the asthma reminds us of the attacks we see occasionally caused by the irritation of a nasal polypus, and which either cease entirely or are sensibly mitigated when the offending polyp is removed. If the direct irritation of the Schneiderian membrane does not, however, determine the asthmatic complication early, we observe the catarrhal state, as attacks recur year by year, to pass downward into the air-tubes, and then, and very often not till then, do we behold the asthma in full force. Under these conditions we must admit bronchial congestion as a most important factor of the asthma.

The causation of summer catarrh has been admirably demonstrated of late years by Dr. Blackley, and I wish that time would allow me to detail some of his experiments. Dr. Blackley finds attacks of hay fever to be most prevalent and severe about the 28th of June, and at this period of the summer he found he could collect on glass slides, moistened with glycerin and exposed to the air, the largest number of pollen granules from flowering grasses.

breath. From that time she became decidedly asthmatic, the attacks commencing with a great itching under the chin, a good deal of coryza and flux, and great tightness across upper part of chest. But little expiratory wheezing was heard after the fit, and great relief was gained by taking moderate doses of hydrate of chloral. In this case, I especially noticed a red erythematous rash, which appeared on the chest whenever the asthma and catarrh came on, indicative, I thought, of vaso-motor disturbance and vascular congestion. The rash did not seem connected with taking the chloral hydrate.

In 1871, I had a medical man, æt. thirty-two, under my care for very troublesome asthma of catarrhal form, and almost always this gentleman observed an eruption of real urticaria to appear on his skin whenever he had his catarrhal asthma. In this case the catarrh preceded by some years the bronchial spasm. The remedy found most useful by this patient was the production of free sweating.

In another case, also seen in 1871, a young man, æt. twenty-one, had been subject to spasmodic asthma, with catarrh, for nine years; and previous to that date he had suffered much from moist eczema on the skin. These instances show the reason there is for admitting vascular disturbance in the way of congestion and hyperæmia as an element in the causation of catarrhal asthma.

The bronchial spasm appears as a secondary neurosis succeeding often to pre-existent catarrh. To attribute the whole of the dyspnœa to congestion alone is what I am not prepared to do; for that mucous congestion intense enough to cause complete obstruction of the nostrils, as in hay catarrh, should extend in like degree by continuity of tissue all down to the smallest bronchi, can hardly be accepted, for surely then larynx, trachea, and bronchi would be so occluded as to cause actual asphyxia.

Asthma due to, or associated with, bronchial congestion, usually gets speedily bad when the patient lies down at night.

A boy, æt. nine, was under my notice in 1873, who never knew anything of asthma till at the age of four, when he passed through an attack of scarlet fever. He had no anasarca, as I was informed, but ever after the scarlet fever he remained liable to asthma, which was always worse when he lay down in bed, was relieved often by sneezing, as well as by

coughing up mucus. The urine was normal, save excess of lithates. The inspiration was attended with a few sonorous sounds. A dry, bracing air did more good than any medicine in this case.

Dr. Blackley, in his observations upon himself when affected with hay catarrh, found that if he lay flat on his back he could easily bring on all the distress of a true asthmatic attack. Blackley considers congestion the sole factor of the asthma. Here, as I need not tell you, I do not agree with him. The congestion causes dyspnœa, but true bronchial asthma requires, for its production, that muscular spasm be added to bronchial congestion.

Illustrations of muscular spasm, induced by contiguity of inflamed membrane, are often brought before us. I may instance spasm of the laryngeal muscles coming on suddenly as a most dangerous complication in laryngitis. Already I have referred to bronchial spasm and asthma as a secondary neurosis resulting from bronchitis; and I may instance the case of a member of our profession, who while travelling about to get rid of a persistent bronchitis, suddenly, while passing a night at Gibraltar, was taken with spasmodic asthma: and so severely did this asthma keep its hold, that when consulting me in August last, this gentleman informed me that for 340 nights out of 365 he had to sit up for two hours or more fighting for breath, "and you may judge what that is," said he, "to a man actively engaged in practice during the whole day." The means by which this physician was eventually much relieved will come before us again presently.

In another case, the course of events tended to a different issue, and a brief narration of the case will bring me to the question of emphysema in connection with asthma.

The patient was a clergyman of ample means, who, in consequence of very troublesome bronchitis, was sent many years ago to Madeira, whence he returned with dysentery as an unwelcome addition to his bronchial inflammation. He told me he knew nothing of asthma till he went to Torquay, and there he was seized quite suddenly at night, and he learned that five people of his acquaintance at Torquay had all experienced severe asthmatic attacks during that same night. The case of this clergyman—referred to, I find, by Salter in his table of cases—was instructive, and illustrative of the course of many

cases of asthma connected with bronchitis. First, the poor man had troublesome bronchial inflammation, which appeared to irritate the nerves from the pulmonary plexus, which Remak has shown to be distributed to the bronchial muscle. Then, under some peculiar atmospheric exciting cause, this muscle was thrown into spasm, and the patient surprised by a fit of asthma; and from that time bronchitic asthma clung to him for the rest of his life. Gradually the spasmodic seizures became less severe, while very marked atrophous emphysema of lungs came on, expiration being notably difficult and prolonged. It seemed, therefore, as if spasm and inflammation together had worn out the strength of the bronchial muscle, and during the latter years of this patient's life, his distress was from over-distended lungs, impeded expiration with paralytic and wasted condition of bronchial muscle.

Few will dispute the proposition that the establishment of an atrophous emphysema of lung materially compromises the permanent recovery of an asthmatic patient. In my last chapter I laid stress on prolonged and laborious expiration as a sign that emphysematous distention of the air-vesicles either was, or soon would be, developed; while, on the other hand, I also stated that where the attack of bronchial spasm was sudden though severe, inspiratory distress marked, the patient young and free from any antecedent inflammation of his bronchi, that then we might look for a complete recovery—a statement confirmed by one or more authorities to whom I make reference.

Now, with respect to emphysema as a concomitant of asthma there is something to be said, for I am sure I have on occasions overestimated the degree of emphysema present in the lungs, and given an unwarrantably bad prognosis of the case in consequence. Some of the latest observations published by Dr. Salter in the "Lancet" make me think he too was conscious of having fallen into the same error. The causes of the mistake I believe to be a want of due recognition of the causes and forms of emphysema, in the first place; and in the second place, want of attention to the way in which the diaphragm is acting. Of the forms of emphysema, one may be a mere mechanical distention of lung, due to impeded expiration; and it is in the lung apices we notice the emphysematous signs first, as these parts suffer first from expiratory pressure.

This was shown in the case of M. Groux, the man who had a congenital fissure in his chest-wall, through which the movement of the lung could be observed. During forced expiration the lung came forward into this sternal fissure as a long tympanitic tumor.

Von Ziemssen, too, had the opportunity of seeing the case of a robust man, æt. forty-five, in whom there existed a congenital deficiency of the pectoralis minor, and of the entire sterno-costal portion of the pectoralis major, so that the muscles of the four upper intercostal spaces were covered only by skin. On quiet inspiration a considerable sinking in was noticed in these situations, which was succeeded in expiration by a filling out of the same spaces; and on forced expiration these intercostal spaces were powerfully puffed out and distended.

When the intercostal muscle was made to contract over the space by galvanism applied, then the bulging was not observed in that one space during expiration.

I have now in Victoria Park Hospital a most typical example of gouty asthma, in a man aged forty years. The depression of the upper intercostal spaces in inspiration is very marked, as if the lung could not follow the expansion of the thoracic wall.

The obstructed expiration due to an attack of bronchitis we know to cause a temporary over-distention of lung—an acute emphysema subsiding with the subsidence of its cause just as the temporary emphysema of whooping-cough subsides as the disease passes away.

Salter says he is convinced that he has seen emphysema of lung come on without any antecedent bronchitis, and in this I agree, though I do not hold that emphysema unconnected with bronchitis is always a mechanical affair and independent of nervous influence. I believe that imperfect innervation through the vagus nerve may lead to defective expiration and emphysema; indeed, experiment has shown that section of the vagus will cause slow breathing, prolonged expiration, and emphysema of the lungs. Emphysema of centric nervous origin may develop with great rapidity, as in a case recorded by Dr. Walshe, where well-marked vesicular emphysema was found in the lungs of an infant that only survived its birth by two hours. No form of bronchitis, pneumonia, or atelectasis

of lung was present, "but," says Walshe, "almost immediately after birth this infant was seized with extreme difficulty of breathing, probably of cerebral origin," and so died.

In a case recorded by my former colleague at Victoria Park Hospital, Dr. Baumler (Path. Trans., XXIII.), where an aneurism pressed on and paralyzed the right pneumogastric, the lungs after death were found large and emphysematous. A former distinguished president and Lettsomian lecturer of this society, Dr. Habershon, has paid attention to the effect of pressure on the pneumogastric nerve, and he observed that first this pressure leads to paroxysmal and spasmodic contraction of the muscles of the larynx; secondly, to diminished muscular power, with paralysis and wasting of the laryngeal muscles; and, thirdly, to pulmonary congestion.

Dr. George Johnson has reported a case of death from an aneurismal tumor pressing on the left pneumogastric and recurrent nerves, and here the lungs were found "very œdematous," and the muscular structures of the larynx notably atrophied.

From these statements we may, I think, infer that emphysema may be developed in the lungs under defective innervation, as well as under the influence of mechanical distention of the air-vesicles.

Provided the nerve failure be not due to any organic cause, these cases of emphysema improve wonderfully often under the influence of a tonic and bracing air, with very moderate elevation above sea-level. A mild, soothing air suits best where intercurrent spasm comes on, and there is reason to suspect a lurking bronchitis. The way in which patients, who have been for months in a constant state of bronchitis and chest oppression, very rapidly improve, so as to be able to run up hills in Cornwall, Ventnor, and other places, makes me believe strongly in defective innervation as a great cause of their emphysema and breath troubles. A suitable climate will often act as a first-rate anti-spasmodic. Under its soothing influence the irritable bronchial muscle gets into regular rhythmical action, and is able to discharge phlegm that has been long collected in the air-tubes.

Time forbids me to quote a few cases that have impressed me much; but when I come to the medicinal treatment of these cases, I shall hope to have time to mention the excellent effects exercised by nux vomica as a medicine for them.

The worst cases are those of atrophous emphysema. Here we find, as in the clergyman's case already reported, a history of bronchitis to weaken the bronchial muscle; next, bronchial spasm to strain and stretch the air-cells; then gradually spasm gives way to paralysis, the lungs waste, air stagnates in them, the blood is badly oxygenated, the heart suffers, and, like the air-cells, becomes either fibroid or fatty, and death by syncope often closes the scene. The clergyman, who was so typical an example of this sad course of events, I was told, died at last of syncope.

These are the cases where expiratory dyspnœa is characteristic and marked. Often I have heard the patients say that they want to get the air out of their chest; and one intelligent man remarked spontaneously that it seemed as if a door existed by which air was let in, but then it would not reopen to let the air out. In this man's case chest distention was well seen, the lower chest not retracted, and the diaphragm obviously depressed.

We are reminded here of those singular cases of asthma described by Wunderlich, where a gradually increasing dyspnœa culminates at its maximum in two or three days, with chest motionless and hyper-resonant with pent-up air, heart and liver both pressed down, the former by distended lung, the latter by distended lung and depressed diaphragm. This condition of chest is a truly distressing one to witness and differs pretty evidently from the severe bronchial asthma, with retracted epigastrium, seen in young persons. If not relieved, the patient becomes livid, and complains of twitching and jumpings in his limbs, due to the circulation of carboniferous blood through the system. The diaphragm here is deeply concerned, being depressed, and apparently in a semi-paralytic condition; and when things are thus, the case is, in my experience, a most unpromising one. That "paralysis of the diaphragm is incompatible with the duration of life," as once stated, is not absolutely true; for Duchenne has clearly proved that life may hold on, without serious disturbance, so long as the accessory muscles of respiration and the lungs remain unaffected, though the diaphragm be, as a matter of demonstration, completely paralyzed. In a work on asthma[2] which I published some years ago I have made reference to this diaphragmatic palsy and inertia as greatly adding to the

intractability of emphysematous asthma; and as experience has grown, I am but confirmed in my belief. I see cases of persons with no great amount of bronchial spasm, who are always short-breathed, have weakness of voice, often tendency to vomiting, both on full and empty stomach, and whose chest and abdomen are distended, prominent, and motionless. When no actual spasm is on, the chest, extra resonant on percussion, with heart, liver, and diaphragm all pressed down, yields hardly any sound when auscultation is applied; little or no air-movement can be heard, and expiration is feeble and long. These are about the most intractable cases one can meet with in practice.

In a few well-marked cases I have noted a sort of expiratory puff, which seems to mark the final act of expiration: the first part of the act is noiseless, but latterly, as the feeble lung contracts on the contained air, this escapes with an audible puff. This phenomenon I have usually observed on the right side of the chest, as if the emphysema was most marked on that side. This reminds us of the statement of Williams (p. 654) that after excessive distention of the lung the bronchial muscle could not be excited to contract till a brief interval had elapsed.

These are the cases in which an irregular zone of congested capillary vessels can often be seen on the chest-wall, closely following the line of attachment of the diaphragm.

As has just been stated, it is to climate we must mainly look for the relief of these cases of over-distended semi-paralytic lung with depressed diaphragm; and I have observed that while often a mild, moist, sedative air is prejudicial to them, they will warmly extol the beneficial action of some climates that are notoriously bad for cases of spasmodic asthma. Thus a man who presented typically the conditions of lung and chest just described, informed me that while staying at Margate he was better in his health than he had been for many months; while at Cheltenham he had been so bad that he could not remain in the place.

In contrast, I would mention more than one case of boys or girls liable to spasmodic asthma who have casually found themselves at Margate for a night, and had there such an attack of spasm in the breathing that they have had to send for the doctor, who has ordered them at once to quit Margate, the air being too exciting for them.

I believe that in this last form of asthma the pure, bracing air of Margate stimulates the bronchial muscle too much and brings on a contractile spasm; while in the former case of the semi-paralytic, over-distended lung the stimulating air rouses the torpid bronchial muscle to more complete contraction, so that stagnant air is expelled from the cells, the blood becomes more freely oxygenated, animal heat is restored, effete tissue eliminated from the blood, and the patient feels warm, hungry, and cheerful.

I am sure I have seen good come also to these highly emphysematous asthmatics from the respirations of diluted oxygen gas, and that, too, in cases where careful trial of Waldenberg's spirometer had failed to give any relief.

Waldenberg's spirometer is an instrument by means of which the patient can expire from his chest into a receiver of rarefied air, for the purpose of thus extracting the stagnant air from his lungs. Secondly, he is enabled by the same instrument to inhale compressed air into the lungs, just as is done by those who pass some time in a chamber or bell of compressed air, and thus to get an extra dose of oxygen. The principle of the machine is ingenious, but my experience of its effect as a means of giving relief in emphysematous asthma is not encouraging.

Galvanization in the course of the phrenic nerve has been suggested, but I have no experience to bring before you as to its success.

A feeble condition of diaphragm has been observed in saturnine asthma, as a result of lead in the system. The importance of recognizing this depressed state of the diaphragm in cases of emphysema, with or without asthmatic complication, I observe to be alluded to in the recent work of Dr. Headlam Greenhow on "Chronic Bronchitis;" and I there learn that thirty years ago Dr. Stokes, of Dublin, drew a distinction between those cases of emphysema, or, as he terms it, dilatation of the air-cells, in which the diaphragm is displaced downward, and those in which it is not so displaced.

Stokes gives the case of a young man, who had distended chest and emphysematous lungs, but yet presented no evidence of depression of the diaphragm. This young man did not suffer from difficulty of breathing in the intervals when he was free from bronchitis, and was able to take very active

exercise; having, shortly before entering the Meath Hospital, walked a distance of forty miles in one day. His chief inconvenience was the frequent occurrence of bronchitic attacks.

In the case given by Dr. Greenhow (p. 253) of a man who made marked improvement from a condition of emphysema, complicated with asthma, it was observed that the abdominal muscles were in unusual activity, and so much hypertrophied that the situation of the linea alba and lineæ transversales was marked by distinct furrows, indicating the powerful nature of the expiratory effort to press up the diaphragm, and empty the lungs of stagnant air.

I trust I have thus exemplified the important part taken by the diaphragm in connection with emphysematous dyspnœa. Till I learned fully to appreciate this, I often wondered why some cases of asthma, not characterized by any well-marked attacks of bronchial spasm, were so much more incurable than other cases, where bronchial spasm was a prominent cause of complaint on the part of the patient; and I soon found that in these intractable cases there was not only evidence of lung-distention and emphysema, but also very imperfect action of the diaphragm, and marked over-fulness of the lower chest. Nux vomica and strychnia, remedies so very useful in pure emphysema, were quite powerless in this condition of diaphragmatic inertia. It is worth while always to seek for a zone of congested vessels, which, as has been said, may be found on the surface corresponding to the line of the diaphragm.

Being desirous in this chapter on the "Pathological Relations of Bronchial Asthma" to say a few words on the true degenerative changes of lung-tissue that may result from prolonged asthma, I am obliged to omit the consideration of the pathological relations of heart disease, renal disease, and gout, and proceed to degeneration of pulmonary tissue.

The alternation of attacks of gout with those of asthma is a fact often brought under our notice. I have known one who, during his youth, suffered frequently from severe asthma entirely to lose the attacks on the development of true gout.

The question before us is, Will prolonged attacks of bronchial spasm lead to phthisical or tubercular disease in the lungs, as well as to emphysematous distention and degeneration of the pulmonary muscles?

Bronchial spasm is a disorder of function; phthisis and emphysema are diseases of tissue, and as a general rule prolonged disorder of function, sooner or later, leads to structural tissue-change. While, as I have already shown, bronchial congestion may become a determining cause of bronchial spasm, so we have the reverse condition often brought to our notice, where severe attacks of bronchial spasm, relieved by inhaling burning nitre-paper, exist for years before any signs of cough or bronchial congestion become apparent. A well-marked example of this course of events I have now under observation.

A man had typical seizure of bronchial spasm for years, and then began to have a cough, and slowly developing symptoms of emphysema and bronchitis as a secondary effect. In seeking into these secondary effects of bronchial spasm, by far the most common is vesicular emphysema of the lungs. Some cases have come under my notice where asthmatic persons have become phthisical, and these have seemed to me examples of the extension of atrophous emphysema into a destructive process involving the lung substance, and so producing fibroid phthisis. For the victim of spasmodic asthma to become really tuberculous, and die with typical gray tubercles present in the lungs, appears a most rare event. One case I have found reported from the German, where a young student, who was liable to most intense attacks of spasm with great drawing in of epigastrium, eventually died; and a post-mortem examination showed the lungs full of gray tubercles. My own experience fails to afford any similar case; and of those that I have noted of patients who have died of genuine acute tuberculosis, I do not find any note of asthmatic attacks as features in their history. In a well-marked case of miliary acute tuberculosis, given by Dr. Peacock, where a young lady, twenty-two years of age, fell ill on May 14th, and died on June 1st, there was dyspnœa, aggravated in the recumbent posture, as a prominent symptom, together with great acceleration of the respirations; but I do not find any antecedent history of asthmatic seizures, and auscultation revealed nothing more than harshness in the respiratory murmur. There seemed no auscultatory evidence of bronchial stricture. Post-mortem—both lungs were more or less airless, and filled with small miliary tubercles, none of which had undergone any softening process.

I do not find any evidence to associate miliary tubercles with attacks of bronchial spasm, though we can conceive the possibility of the conjunction of the two affections in certain cases, from what we know of the causation of bronchial spasm by bronchitis and bronchial congestion.

That protracted asthma should lead to a phthisical destruction of lung, is not a common event, and yet I can find illustrations of this occurrence from my own experience, as well as from that of others.

In Salter's table of 223 cases of asthma, there appear 49 in whom there is a distinct account of emphysema of lungs and asthma existing in some of the members of the patient's family. In 14 cases we find history of phthisis as an hereditary disease affecting parents, brothers, sisters, uncles, or aunts, and in about half of these 14 cases the physical signs as recorded were such as would suggest the possibility of phthisis, but I only find one mention of hæmoptysis among them.

Looking over my own notes of cases where asthma has issued in phthisis, I find the termination certainly an uncommon one, and I find hæmoptysis nearly always recorded among the symptoms.

The cases prone to go into phthisis are those of persons approaching to, or fairly in, middle life, who have been for some time asthmatic, who come of an ancestry liable to pulmonary disease, and in whom the physical signs of atrophous emphysema of lungs are well marked.

The presence of small fibrinous casts of the minute bronchi in the expectoration always makes me expect a phthisical termination of the case, and there was in our museum at Victoria Park Hospital a most characteristic fibrinous branching cast of some of the twigs of the bronchial tree that had been coughed up by a gentleman, who, having been for many years liable to asthma, eventually died of pulmonary phthisis.

In atrophous emphysema there is feeble nutrition of the coats of the bronchi, and the fibrinous casts formed tend to irritate and destroy the weak bronchial membrane, and so to set up ulcerative destruction of the lung. A natural inherited weakness of the bronchial membrane and air-vesicles may constitute the hereditary element in the case, and such a weakness may be derived from parents affected with any form of pulmonary disease. Of three cases where I was able to get

full and complete notes of asthma turning to phthisis, in one the fibrinous sputum was noted, and this was in the case of a man who died late in life, and when young had suffered much from gout.

Of my two other cases, one was that of a gentleman, æt. sixty, who for years was liable to very severe spasmodic asthma; gradually spasm passed off, dyspnœa became constant and abiding, blood appeared at intervals in the expectoration, moist crepitation became distinct at right lung apex. Temporary benefit followed on the use of cod-liver oil and hypophosphite of soda, but in about three years death, with all the signs of phthisis, took place.

My second case was that of a lady, æt. twenty-one, whose father was living, and a great sufferer from bronchial asthma. The daughter, during her childhood and early youth, had also suffered severely with fits of asthma, usually relieved by inhaling the smoke of burning nitre-paper. When I saw her in September, 1876, these spasmodic attacks had passed away, but she had a pulse of 120, copious purulent expectoration, at times streaked with blood, and abundant sub-crepitant râles over upper third of left lung, and hoarseness of voice. She improved more than I expected on pills of creosote and syrup of hypophosphite of lime; but rather more than twelve months after I first saw her, I heard of her death from phthisis at her home in the country.

CHAPTER III.

TREATMENT OF ASTHMA.

Having in the two preceding chapters endeavored from the observation and history of cases to point out what distinctions we may recognize among the various forms of bronchial asthma, I now seek to show how far the correct appreciation of these distinctions can be made available to guide us in the choice and application of our remedial measures.

Floyer, writing in 1717, says: "Since the cure of the asthma is observed by all physicians who have attempted the eradicating that chronical distemper, to be very difficult and frequently unsuccessful, I may thence infer that either the nature of that disease is not thoroughly understood by them, or they have not yet found out the medicines by which the cure may be effected." Now, if physicians have not yet found out the medicine to cure asthma, it certainly is not for want of experimenting with various drugs and preparations; for there seems hardly a known medicine that has not been tried, and I could enumerate a long list for which distinguished success has been claimed by their respective advocates.

Further to pursue the plan of specific-hunting is hardly likely to advance us much toward what is positive and practical, and the more promising course appears to be to seek to turn to account the knowledge we have gained by experiment and observation of the nature and causation of bronchial spasm and congestion. Thus we may discover how to select and apply some of those remedial means with which we are so amply provided.

Experiment has demonstrated how much respiratory action is under the influence of the pneumogastric nerve, and reflection on the wandering course and numerous connections of this nerve will show why it is that our treatment of asthmatic spasms must vary, and is not always so precise in its results as we could desire.

To cure asthma we often have to treat other organs than those of respiration, and the way to success may lie in the correct adjustment of a displaced womb, in the administration of a dose of extract of male fern to dislodge a tapeworm, or in a course of Carlsbad waters to clear the bile-ducts of one who perhaps for long has been smoking all manner of cigarettes with little further effect than the production of vertigo and giddiness. I could illustrate each of these conditions by a well-marked example.

The capriciousness of asthma with regard to the influence of various agencies is notorious, and a book might be filled with an interesting collection of cases.

What better illustration can I find than a glance at the effect of atmosphere and climate on persons liable to asthmatic seizures? Theory would say, it is very clear that, seeing how bronchial spasm and air-stoppage must have the effect of carbonizing the blood, a pure air is the first requisite for an asthmatic patient. Practice confirms this often in cases of congestive asthma with want of tone in vessels; but to the victims of true spasmodic asthma a pure, clear air is often utterly unbearable, and we find them hurrying away from the bracing air of places like Margate, Whitby, and Nice, almost in terror of losing their lives if they pass another night in one of those climates.

The late eminent oculist, Mr. Dalrymple, of Grosvenor Street, when summoned into the country, was not uncommonly compelled to hurry back to London without even seeing his patient, in consequence of the terrible spasm in his breathing that came on in the pure air of the country.

Salter has gone carefully into the history of 20 cases of asthma powerfully influenced by atmosphere; the evidence adduced is of a most opposing character, and of little practical avail. Fourteen out of these 20 cases could respire with comfort only in the smoky air of populous cities; 11 seemed actually cured by London air; 7 others were quite unable to live in cities, and did best in the country. From the perusal of Salter's reports, as well as my own, I cannot find, either in peculiarity of patient or of atmosphere, the cause of this capriciousness in the way the asthma selects its victims. If it be the dryness of London air that is so wholesome for asthmatic lungs, how comes it that Lord B., as

told us by Dr. Ogier Ward, has been obliged to leave London at two o'clock in the morning and drive to Epsom, the intensity of his asthma threatening his life? At Epsom he is said to find immediate ease, and yet the air there is reputed a dry air.

The wonderful relief to breath difficulty experienced by many asthmatics in the close air of a town is continually brought to our notice. One patient of my own, with a most comfortable home in the northern outskirts of London, finds himself glad to quit it and pass a winter in residence at his business premises in Cornhill. Another, who declares that he has vainly tried all the resources of medical art, passed six weeks in Queen Anne Street, Cavendish Square, without feeling a trace of asthma. On going to Surbiton he got such a bad seizure, that though well versed by sad experience in his sensations, he was nevertheless obliged to send urgently in the night for the doctor. One great sufferer in the country breathed well in London, but wrote me word that on returning home the asthma received him with a most warm welcome. Those who have read Walshe's work on the lungs will remember the case of the man sorely tormented with asthma at Hampstead, for whose relief every known remedy had been vainly tried, and who was cured in an almost miraculous way by changing his residence to the central region of the Seven Dials. To explain why town air suits some asthmatics so well is not easy. Finding that ozone inhaled causes pulmonary irritation, I thought that it might be the absence of ozone that rendered the air of towns sedative to excited nerves in the respiratory passages.

In connection with this question I may refer to the observation of Schönbein, the discoverer of ozone, who, in his experiments, found that air highly charged with ozone when inhaled brought on a painful affection of the chest—a sort of asthma with a violent cough, which obliged him to discontinue for a time his investigations. Struck with this, Schönbein got several physicians at Basle to compare their lists of catarrhal patients with his table of atmospheric ozonometric observation, and he and they were struck by the unusual prevalence of catarrh on the days when the iodine test-papers showed that ozone was unusually abundant in the air. The observations of Dr. Carl Haller, of Vienna, prove that catarrhs and pulmonary inflammations rise and fall in frequency, very

much in the ratio of the presence or absence of ozone in the air.

Schubler and others experimenting on animals (see Dobell's third volume of "Reports on Chest Diseases") found that ozone inhaled had an irritating effect on the respiratory organs somewhat resembling the action of chlorine gas. After breathing ozonized air for five or fifteen minutes, catarrhal irritation of the eyes and air-tubes came on, the respirations became slow and difficult, and in two or three hours death took place. Post-mortem examination disclosed œdema, and in some cases inflammation, of the lungs. The discomfort sometimes experienced by asthmatical subjects on a sunny, bright day in the neighborhood of luxuriant vegetation, may be due to the ozone given off by growing plants when exposed to the action of sunshine. That, however, ozone in the air is not the invariable cause of asthma, is shown by the following facts:

Dr. Blackley found that he could expose himself to sea air highly charged with ozone, as evidenced by the iodine test-papers, and remain perfectly free from anything like asthma and catarrh. A young and bad asthmatic among my own patients was always in great respiratory distress on the sea-coast, and naturally dreaded a voyage to Australia. He embarked at Gravesend, and while the ship was going down the Thames he was obliged to resort to his pipe; but once fairly at sea he had no breath difficulty whatever, and wrote to me from Sydney to say the sea-voyage had cured his asthma in a way that greatly puzzled him, his own sensations leading him to the impression that the phosphorescence of the sea-waves had much to do with the promotion of free respiration. At any rate, the experience of this young man showed that seaside air may be an effective cause of bronchial asthma in one who, fairly out on the ocean in a ship, loses every trace of his troublesome disorder. Careful ozonometric observations made during the voyage from Lizard Point to Cape Otway (Victoria), showed that ozone was always present in the air, the amount ranging as from 10° to 3°. If ozone be always in all cases an exciting cause of asthma, one could hardly expect a person eminently subject to the disease to make the voyage to Australia without the least respiratory distress.

I have searched for evidence of asthmatical people becom-

ing attacked after a thunder-storm, when the air is full of ozone. Some there are who are uncomfortable in this sort of air, and among Salter's cases one is mentioned who had asthma complicated with bronchitis, and who was, after a cessation of spasm for some years, attacked unexpectedly and severely after a thunder-storm. When this attack had passed away there was no return for a period of five years.

The facts thus narrated seem to me worth bearing in mind, in hope that increased experience and observation may lead to something more positive in the way of a result than we have a right at present to claim, for as yet we are obliged to say that the climatic treatment of asthma is an experimental affair, and the best practical rules we can lay down appear to be that, if spasm be very pronounced, then probably a town air will suit best. If the attacks are associated with atony of system and loss of vascular tone, a dry, bracing air seems to me to do most good ; and, at all events, if a patient be very bad on a certain soil, or in a certain kind of air, get him away to a locality and air of as different a nature as can be found. A mild, sedative climate, like that of Hastings, Ventnor, or Torquay, will do wonders, I find, for some patients, who suffer greatly with chronic bronchitis, pulmonary emphysema, and bronchial spasm, when in town. The mild air soothes the irritable bronchial muscle, and, causing its free and rhythmical action, allows a more complete aëration of the blood with easy expectoration, and then increase of strength and appetite soon follows.

In selecting for consideration some of the numerous means employed for the relief and cure of asthma, I speak first of the use of certain fuming inhalations that experience has proved valuable in alleviating bronchial spasm. The well-known nitre-paper is best made by dissolving 4 oz. of nitrate of potash in a pint of hot water, and in this should be soaked a porous paper of the thickness and consistence of ordinary blotting-paper. The paper thus made will be a strong, fiercely burning paper, and should be kept in a stoppered bottle. When used it must be burnt fast and furiously, so as to fill the room, and commonly it is not till the atmosphere is quite thick with nitrous vapors that the asthmatic gets relief. On this point experience leads me to speak strongly. Salter used to say that the more purely spasmodic the asthmatic attack,

the more speedy and certain became the relief obtained from the burning nitre-paper, and that if any bronchitis was present, then the nitrous fumes might even add to irritation and distress. There is some truth, I am sure, in this statement; nevertheless, a physician well able to judge, declares that in his asthma and bronchitic cough the fumes of the nitre-paper are more effectual than any other remedy in affording relief. The sort of case to be treated by nitrous fumes is such a one as this: Miss W., æt. twenty, had asthma since her fourth year. The attacks came and went with great suddenness—evidence, I take it, of their spasmodic nature—and her father, who was himself a medical man, obtained all sorts of advice for her, to no purpose; though, when sent to the seaside, she lost her asthma for four months. One morning the state of things stood thus: the young lady was sitting with body thrown forward, shoulders raised, and the noise of the air straining through the constricted tubes so loud as to be heard in the lower part of the house. A sheet of nitre-paper was burnt till the room was filled with a cloud more dense than even a London fog. Scarcely had two minutes passed when, changing her position, she lay back on her pillows, and in ten minutes her delighted father found her breathing as quietly and noiselessly as an infant.

Early one Sunday morning in March, 1870, Dr. Salter, being himself indisposed, asked me to go down into Kent to see a bad case of asthma, to which he had been urgently called. When I got to the patient's house I found him sitting in his drawing-room, feeling pretty comfortable; but his doctor told me that it was not till he had regularly filled the room with a suffocating vapor of burning nitre-paper that any relief to the breath was obtained.

This gentleman was sixty years old, and had been asthmatical from the age of twenty. About that time he told me that he consulted a then eminent physician in Finsbury Square, who, as soon as he saw him, and had heard a word or two of his story, said:

"Little use your coming to me, young man, for I have had asthma all my life, and have it still; if there is anything to do you good, it is strong coffee."

This patient lost his asthma while at Ventnor. When he got to his own home, near a river, back it came again, and the

fits grew upon him till he became almost black in the face. Dense nitrous fumes surpassed anything else in giving ease to his breathing.

Though experience has taught me the necessity of pretty well smothering a bad asthmatic with the nitrous fumes, still I have come across cases where a small inhalation of a mild nitre-paper appears to prevent asthma attaining a climax of severity. The sleeping patient turns and becomes uneasy in his respiration, and then nitre-paper burnt near to him may have the happy effect of easing his breath and maintaining a condition of calm repose; if no one is at hand to light the paper, the fit culminates in a wakeful spasm of severe kind, requiring chloroform vapor for its relief.

I cannot ascertain who it was that discovered the value of burning nitre-paper to cure the asthma. In the "London Medical Gazette" of September 4th, 1846, there is an article extolling the remedy very highly; and we learn, in the "Lancet" of April 5th, 1845, how a friend of Mr. Harrison's tried burning blotting-paper that had been soaked in a saturated solution of nitrate of potash, and said he felt the inhaled smoke to clear the passages and open the air-tubes; and though he had used this remedy twenty times he had never been disappointed in finding relief in fifteen or twenty minutes.

In America there is a story that an asthmatic man in great distress took out his pipe to smoke, and found he had no tobacco. Feeling that smoke something he must, he lighted his match-paper, and stuffed that in the bowl of his pipe. The paper fumed away, and he, as he inhaled the smoke, found it a quicker and better cure than the tobacco had hitherto been.

In Salter's table of 223 cases of asthma, there were 65 to whom nitre-paper proved a most valuable remedial agent. Several of these are noted as having emphysema and slight bronchitis to complicate their asthmatical spasm, showing that a slight degree of these two conditions does not invalidate the remedial action of the paper.

The chemical products resulting from the combustion of nitre-paper have been examined by M. Vohl (see "Journal de Pharmacie et de Chimie," 1866, p. 155), and he found the nitrous fumes to contain watery vapor, traces of cyanogen, carbonic acid and nitrogen gases, ammonia, and nitrate of potash. To the ammonia and nitrate of potash, M. Vohl attributes the

anti-spasmodic action of the nitrous fume. The constituents of the fume seem so closely allied to the adventitious impurities found in the air of smoky towns, that I went over Salter's table of his 223 asthmatics, to see if I could make out that those persons who are put down in the table as being notably relieved by the nitrous fume. were also those who were either much relieved or quite cured by London air. I found that of the 22 who are reported to have obtained marked relief to their breath from the air of London, there were 9 to whom the nitre-paper was very valuable. Two, who are said to have been quite cured by London air, found the paper more serviceable than anything else they had tried. One, who suffered much in London, found nitre-paper rather harmful than otherwise. It is to the ammonia and carbonic-acid gas that I think we may attribute some of the anti-spasmodic action of the burning paper, for it has been shown by Trousseau and others that often ammonia vapor will speedily cut short an attack of bronchial spasm ; and Salter reports one case where the spasm was at once nipped in the bud by cautiously inhaling the vapor of diluted ammonia. An asthmatic sea-captain was free from his breath trouble when conveying a cargo of guano, a product which slowly evolves ammonia. To carbonic-acid gas some degree of anti-spasmodic power has been attributed; for it has been stated that the reason why an asthmatic, when black in the face, and apparently at the very verge of asphyxia, suddenly begins to mend, is that the carbonic acid in the blood acts as an anti-spasmodic and sedative to the nerves of the bronchial muscle, and so relaxes the spasm —a theoretical notion which as yet does not seem to me convincingly demonstrated.

That the nitre-paper fume is an anti-spasmodic I judge from the very speedy way in which it eases sudden seizures of breath difficulty. A man whom I saw a few days ago with extensive heart disease and dropsy, but no bronchial spasm, had his symptoms commence with sudden asthmatical seizures, the spasm coming on in such force that he had to leap out of bed in fear of suffocation. Burning nitre-paper gave relief speedily ; but he was glad to give up his practice of now and then smoking his pipe, for tobacco brought on faintness and great depression. Among Salter's tabulated cases I observe how frequently those who were relieved by the nitre-paper

found good also from the use of a pill containing ½ grain of extract of stramonium.

Nitrate of potash forms, I expect, the basis of the numerous list of proprietary papers sold for the relief of asthmatic persons. Some patients fancy one kind of paper, some another, and very many think nothing can surpass their own home-made nitre-paper. I have made and tried papers containing iodide of potassium, as well as nitrate of potash and chlorate of potash, but do not find them to be in any way superior to the nitre-paper. The well-known "ozone-paper" of Huggins contains iodide of potassium, as well as nitre, and is a most useful paper. Nitrate of ammonia added to the nitrate of potash appears to have no further effect than to diminish the activity of the combustion, while chlorate of potash increases it.

Dr. Robert Bree tried stramonium smoke in 82 asthmatic cases: in 58 it had no permanent effect, and in the remaining 24 it acted injuriously. General Gent, who first introduced this practice of smoking stramonium for the cure of asthma, himself died from the over-use of his specific. The datura tatula seems a safer and better herb for smoking than the datura stramonium, and in asthma complicated with slight bronchitis I have known the datura tatula smoke prove very soothing. In common with many of my friends, I have had my attention drawn to a variety of powders which, when ignited, burn and give off an anti-spasmodic smoke, and it is right to say that the powders of which I have had experience, known as Senier's, Himrod's, Clery's, are really valuable remedial agents. A patient of mine at Victoria Park Hospital carries some of Senier's green powder in a tin box, and if taken in the street suddenly with spasm in her breathing, retires to a corner, puts some powder on the lid of the box, and lights it with a match; in a minute or two her breathing is relieved. Another hospital patient carries always some nitre-paper of his own make, and uses it in a similar way, and with an equally satisfactory result. I have myself soaked nitre-paper in tinctures of stramonium and verbascum, having seen the verbascum thapsus, or great mullein, recommended in the treatment of asthma; but the objection I find to the use of papers thus medicated is that their fume, when burning, while it relieves the asthmatic breathing, is apt often to produce an unpleasant headache afterward. I prefer to let the patient

try simple nitre-paper first, and if that fails he may sprinkle on it some of one of the powders just named, or dry powder of stramonium leaves and seeds, or of dry belladonna leaves, and see if such addition prove of service to him.

Speaking of anti-spasmodic inhalations, I should like to say a word or two on the use of arsenious acid. That the vapor of arsenious acid has a special action on the respiratory organs seems a well-established fact; and I am informed, on good authority, that men who work in the Cornish arsenic works are seldom affected with phthisis. Indeed, in one instance brought to my knowledge, all the symptoms of phthisis seemed to be removed by exposure to the vapor of the arsenic subliming flues. Whether this happy result was due to the antiseptic action of arsenic vapor, or to some special action of the same on the nerves of nutrition, I cannot say. The fact reminds us that the smoking of arsenical cigarettes has been highly commended by good authority in the treatment of chronic phthisis.

There are various formulæ given for preparing the arsenical cigarettes, known as cigarettes of Dioscorides, of Boudin, and of Le Vasseur. Trousseau recommended that each cigarette should contain 1 grain of arsenite of potash—rather too strong a dose, I think, for a few full inhalations of a cigarette containing half, or less than half, this amount of arsenite will usually answer the desired purpose well.

It is well known how, in Styria, the peasants eat small quantities of white arsenic to improve the breathing powers, and with the British public the use of arsenical inhalation to relieve asthma is not unknown. Dr. Wilks told me of a former out-patient of his at Guy's Hospital who, in his occupation as a stuffer of birds and animals, used much white arsenic. This man was a smoker, and very liable to asthma, and he said he always found a ready cure for his breath-spasm by adding a little arsenic to the contents of his tobacco-pipe.

Of the inhalation of nitrite of amyl in asthma I have not much to say. I have used it, and prefer to employ chloroform, as being more certain, more agreeable, and perhaps more safe. The nitrite varies much in strength, and acts with corresponding uncertainty on the patient. The best way to keep it is in hermetically sealed capsules. So far as my present experience of iodic ether or iodide of ethyl goes, it is encouraging,

and, as this is a new remedy, I will give what report I can of its effects. Pure iodide of ethyl, (C_2H_5) I, is prepared by mixing amorphous phosphorus with absolute alcohol, and then adding iodine. After a few hours the process is complete, and the iodide of ethyl can be separated by distillation; alcohol and iodide of phosphorus producing iodide of ethyl, phosphoric acid, and water. The iodide so prepared should be almost colorless, and must be kept in a dark place, or it will become decomposed and of a brown color from the separation of free iodine. My late clinical assistant at Victoria Park Hospital, Mr. Macdonald, has been good enough to provide me with the notes of three cases, under Dr. Andrew and myself, in which the iodide of ethyl was tried. I will add a summary of these notes:

Case I.—C. D., æt. forty-two, admitted November 19th, 1878; asthmatical five years; has an attack every morning at five A.M., which lasts for about three hours, then passes off, and leaves him free from dyspnœa till the following morning.

November 23d.—Ordered to inhale 10 drops of iodide of ethyl four times in the day. The effect was diminished expectoration, and what is coughed up is white, having formerly been of a greenish hue. He said on December 12th that his attacks were neither so severe nor so frequent as they had been before he used the iodide. The urine and sputa when tested gave decided evidence of iodine.

Case II.—Elizabeth S., æt. seventeen, had been under my observation at intervals for two years, and has suffered severely with attacks of spasmodic asthma and frequent cough and expectoration. Twelve months ago she improved greatly and gained weight under the use of small doses of arsenical solution.

December 10th, 1878.—Has come from Lincolnshire into Victoria Park Hospital, and every morning till December 14th she had at five A.M. a bad attack of spasmodic asthma, with tightness and wheezing of the chest. Strong coffee relieves her, and during the day she is to all appearance perfectly well.

December 14th.—Began to use 10 drops of iodide of ethyl four times a day. She used it thus for one week and then left it off, having had no asthma; and when she quitted the hospital she had been three weeks without needing the iodide; she

had not a trace of dyspnœa even after meals, no cough, and no expectoration.

In the third case, that of a man with bronchitic asthma of seventeen years' standing, the iodide, deeply inhaled, caused a temporary sensation of vertigo and dizziness; the breath, however, was relieved, and expectoration promoted.

A patient, who for twenty years had suffered much from asthma and bronchitis, told me he attributed a great improvement in his case to the use of iodide of ethyl for three weeks. "I think" (writes he) "it tended to discuss or lessen the swollen condition of the bronchial tubes; my doctor wishes me to continue it, but being a new remedy I am rather afraid, though it gives me more permanent relief than anything else I have tried."

The iodide of ethyl seems to be anti-spasmodic, and further, an anti-congestive remedy, allied in action to iodide of potassium, a salt which, when freely and perseveringly given, is well known to be a trustworthy remedy in congestive asthma.

Iodide of potassium is a medicine often of such well-marked efficacy in relieving much of the distress and discomfort attendant upon asthmatic seizures, especially when of a congestive character, that a word may well be said as to the indications for its employment, and the method of its administration. In the case of persons of a rheumatic constitution, and whose breathing is greatly oppressed in damp weather, the iodide is decidedly worth a trial, and it may be given in a commencing dose of 5 grams combined with the carbonate of ammonia mixed in plain water. When the expectoration is scanty and glairy this form appears to suit well; but often it has to be taken perseveringly for as long as ten or fourteen days before its beneficial action is manifest.

With some persons a very small dose of iodide of potassium causes languor and depression, with pain over the eyes and headache, and for such some other medicine must be found. Iodide of potassium is seldom a good medicine for young children under two years of age. Remarkably good results in the way of curing congestive asthma, complicated with bad attacks of spasm, may be obtained by causing the patient to take the iodide of potassium with ammonia three times in the day, while he takes every night a pill containing $\frac{1}{2}$ grain of

extract of belladonna. The belladonna appears to quicken the circulation through the capillaries of the lungs, and certainly aids the iodide in removing bronchial congestion of passive form. Sometimes the belladonna has to be pushed to the extent of producing some dryness of the throat before its full therapeutic action is brought out.

A patient undergoing this method of treatment will also be helped much by the inhalation of 10 drops of iodide of ethyl from a piece of lint whenever he feels the spasm coming on with severity, for it is always best to try and stop the spasmodic seizures at their onset.

A most valuable, and, as far as my twenty years' experience goes, a perfectly safe internal medicine, both for spasmodic and congestive asthma, is found in the various preparations of arsenic. Arsenious acid in a dose of 1-30th of a grain, or the liquor arsenicalis in doses of 2 to 5 drops in water, may be given three times in the day after meals.

Comparative experiments make me prefer the arseniate of soda, a salt which is best given in the form of 3 to 5 minims of the liquor sodæ arseniatis. This dose, given in water, or infusion of calumba, I have found wonderfully efficacious in curing nocturnal attacks of bronchial spasm, complicated very often with obstinate chronic bronchitis and yellow thick expectoration. Where inflammatory symptoms and feverishness are present, or where the heart is feeble, preparations of arsenic are best withheld. Blood-spitting, too, is decidedly against the administration of any form of arsenical medicine. It is well known that the Mont Dore and Bourboule waters, of such high repute in the treatment of the asthma, owe their virtue mainly to a small proportion of the arseniate of sodium. In catarrhal asthma, as well as in the dry form of the disease, the Bourboule water has proved eminently curative. According to careful analysis, by M. Millot, la Grande Source Perrière de la Bourboule contains a constant quantity of arsenic in the porportion of 7 milligrammes to the litre (1 milligramme = about 1-70th of a grain, 1 litre = 35¼ fl. oz.), so that much good may result from what appears a very minute dose of arsenic.

In cases of asthma associated with genuine gout, or with osteoid arthritis, arsenical preparations should be always tried. They will often agree well, and do not run the strength away

as iodide of potassium does when long and largely employed in these cases.

I am compelled to pass briefly over the remedial powers of hydrate of chloral, a speedily palliative agent in most forms of spasmodic asthma uncomplicated with any form of organic disease, and where the respirations are noticed to be very hurried and quick. Syrup of chloral in dose of ½ to 1 drachm in camphor water is often of great temporary service to us, but its habitual employment leads to languid action of the heart and great depression of the system; and if the patient have any kidney disease, symptoms of uræmic poisoning may be brought on by the chloral.

I pass on now to speak of citrate of caffeine, a safe and useful anti-spasmodic, lately brought prominently forward as a curative agent in asthma, and acting like belladonna, and unlike opium, as a quickener of respiratory action. The citrate of caffeine is a white salt, crystallizing in soft silky crystals, soluble in water, and for more than twenty years it has been occasionally prescribed in France and America for the relief of neuralgia and sick headache. Quite recently, I see by an article in the "Practitioner" for January, 1879, that Dr. Lewis Shapter, of Exeter, has been using the citrate of caffeine in 3-grain doses with great success as a diuretic in cases of cardiac dropsy.

I am indebted to a distinguished member of our profession, in a northern town, for example notes of the effect of the citrate of caffeine. I have already referred to this case in Chapter II. For 340 nights out of 365, the patient had to sit up, struggling for breath, and the effect of ordinary remedies may be thus briefly given: burning nitre-paper useless, or worse than useless; tobacco or stramonium, smoked *ad nauseam*, slight benefit; chloroform inhalation, transient relief: nitrite of amyl, tried repeatedly and carefully, entirely useless: phosphorus, arsenic, iodide of potassium, no benefit whatever. After this utter failure of all ordinary remedies, it occurred to the patient himself to try the citrate of caffeine, seeing that the use of strong infusion of coffee always gave him some amount of relief. One or two grains of citrate of caffeine were taken at bedtime, dissolved in a cup of coffee; and if, during the night, asthma seemed about to come on, then an extra grain was at once taken. The effect of this plan of

excess of carboniferous and albuminoid matters that are oppressing it, and the renal organs will also eliminate excess of effete material from the blood; and as this takes place a feeling of relief and elasticity will be experienced by the patient eminently conducive to the removal of his asthmatic troubles.

One of the abbots of the Monastery of La Trappe was entirely cured of a most obstinate asthma by the rigid diet of his brotherhood, involving complete abstinence from meat.

A congested and plethoric state of the vessels of the abdominal viscera can be, to a great extent, removed by a steady course of the Carlsbad or Friedrichshall water, aided now and then by a small dose of mercurial pill; by these means the diaphragm is greatly helped in its action, and free respiration promoted.

The abdominal functions being thus regulated by attention to diet and the use of medicines appropriate for the purpose, if we still find the asthmatic breathing very distressing, the chest distended, the heart's dulness obscured, and that organ's impulse very perceptible at the epigastrium in consequence of its lowered position in the chest, then we may use small doses of the tincture or extract of nux vomica as a promising medicine. Great prolongation of expiration I have usually found an indication for this remedy. This indication for the use of the nux vomica was well illustrated by an interesting case recorded in the "Dublin Quarterly Journal," May, 1860, by Dr. J. F. Duncan.

Eliza Simpson, a married woman, forty years old, had a hard cough, frothy expectoration, and intense dyspnœa. She attributed her sufferings to a cold caught in the snow twelve months previously. The chest was of a rounded form, hyperresonant on percussion, and loud, sonorous, cooing râles were audible in every direction. On inspiration there was no true expansion, the whole thorax moving up and down as if ribs and cartilages were united into one compact structure. The expiration, as measured by hand and stethoscope, was judged to be three times as long as was the inspiration. For treatment a variety of expectorants were tried without success, though a mixture containing nitric acid seemed to agree, and to be of some little service.

December 26th.—A current of electricity was passed

through the chest from the side of the neck to the pit of the stomach, but was productive of no benefit.

December 27th.—She was ordered three times daily a pill containing ½ grain of extract of nux vomica and 1 grain of ipecacuanha. The effect of this pill was surprising and unexpected; she was able to lie down and sleep all the night, and continued to improve steadily. The pills kept the bowels regularly open, promoted expectoration, increased appetite, and procured sleep. Auscultation showed a gradual loosening of the bronchitic râles, and the expiratory murmur was less prolonged.

January 6th.—She left the hospital perfectly recovered. The opinion expressed here was that protracted inflammation had gradually induced a weak and paralytic condition of the bronchial muscle; hence the very prolonged expiration. The breathing appears to have been very early and severely affected. Probably these early attacks, some twelve months before the patient came under observation, were of a true spasmodic nature; and by degrees, as the muscle became worn out by prolonged irritation, the spasm gave way to paralysis. There could not have been any actual degeneration of muscle, seeing how rapid and complete was the recovery under the influence of a nerve-tonic medicine.

As has already been stated, it is probable that in many of these cases of paralytic dyspnœa with overfull and distended lungs, and prolonged expiratory breath-sound, the paralytic condition is due to failure of power in the vagus nerve rather than to degenerative change in the bronchial muscle and air-vesicles. In the case of such a patient as the one just noted, who was but forty years old, it is quite possible that defective power in the vagus nerve may have been one cause of the paralytic dyspnœa. Such cases may be regarded very hopefully, for they usually improve under the influence of small doses of extract of nux vomica, or of strychnia, in combination with nitric or phosphoirc acid.

In the paralytic dyspnœa due to an atrophous emphysema of lung in one who is advanced in life, the prognosis is by no means so favorable, for such a patent may go into a phthisis with destruction of lung substance, or the heart may participate in the same degenerative processes which have involved

the air-cells and bronchial muscles, and the patient may die from exhaustion of heart-power. In such a case no remedies have seemed to me better than hypophosphite of soda or lime, alternated with tincture of iron and liquor strychniæ. Arsenical preparations also are of some service, provided they do not disturb the action of the heart.

It may not be out of place to enlarge somewhat on the treatment of cardiac asthma. Cases of this disorder are recognized, when confirmed, by the gasping character of the dyspnœa, and its tendency to come on after the slightest exertion, or after any exposure to sudden cold. There is more or less of a livid, congestive look about the patient, the feet may be œdematous, and the urine scanty, thick, and high-colored. The respiratory murmur is usually of a harsh character, with little or no sign of prolonged wheezy expiration. The heart's impulse is diffuse, often irregular, perceptible by finger at epigastrium, and on listening the first sound is dull and toneless.

If a patient of this class has been taking abundance of highly nitrogenous aliment in the form of mutton chops and extract of beef, with a fair supply of alcohol, it may be well to begin the treatment by stopping some of those good things, for possibly the blood is overloaded with the products of imperfect assimilation, and such a condition of things, commonly spoken of as suppressed gout, most undoubtedly increases the irritability of the cardiac muscle, and before long will add renal disease and granular kidneys to the patient's already sufficient list of troubles. Rusks soaked in milk for breakfast, and a light dinner in the middle of the day of fish, chicken, or game, with some very weak brandy or whiskey with water, and a cup of milk, or some sandwiches of meat and bread at bedtime, after a light meal about six in the evening, will be a good change. At the same time an alkaline tonic, or an alkaline saline aperient, will aid to relieve the kidneys and liver, and after a week or so the urine will be clearer, the appetite stronger, and the nights much less uncomfortable.

If now we find the heart very feeble and irregular in action, the infusion of digitalis, in doses of 1 or 2 drachms three times a day, will often improve the breathing in a remarkable way. Nux vomica, strychnia, and iron, are also of use; but the effect of the first two is not so marked in cardiac asthma as it is in the paralytic dyspnœa recently described.

In treating cardiac asthma it is important that the patient take small meals at frequent intervals, and it is best to avoid the use of much fluid aliment.

It is in cases of confirmed asthma with emphysema of lungs and weak dilated heart, where so much benefit is said to be obtained from the use of compressed-air inhalations. My own experience of this method of treatment is not large; but in Paris, Frankfort, and other places on the Continent, the system may now be tried under a very perfect management of the apparatus: and from what I have observed of patients who have made trial of the compressed-air bath, it seems of service to persons decidedly emphymatous in the lungs, and who suffer from coldness of the extremities and over-fulness of the venous system. In one instance the effect of the bath on an elderly gentleman, whose case presented the above-described symptoms, was to produce a very free expectoration, with great subsequent ease to his respiration. At the same time a young patient of my own, who had been recommended to try the bath for pure spasmodic asthma, assured me that after twenty or thirty baths he was in no way relieved.

Independently of the effect of increased pressure on the surface, there is no doubt but that by breathing the compressed air a larger quantity of oxygen is conveyed to the blood; hence the remarkable efficacy of these compressed-air baths in curing cases of very obstinate anæmia. (See Report of the Compressed Air Bath in "Notes on Asthma," third edition, p. 150.)

In those numerous instances where difficulty of breathing is made worse by the recumbent posture, on account of the pressure of the abdominal viscera upward against the diaphragm, and where necessity often compels the prolonged maintenance of an upright position till the patient is quite exhausted, very great relief can be gained by the use of Dr. George French's respiratory brace. This contrivance consists of a cross-bar, from the extremities of which hang two loops of strong elastic webbing for the support of the shoulders. A broad band encircles the head, and is steadied by guys stretching across on both sides to the upright elastic supports. The apparatus is suspended by a pulley or ring from the ceiling. When the patient is tired of suspension by the shoulders, he can easily shift his position, and rest his elbows in the elastic

loops or slings. By saving muscular effort to the asthmatic patient, sleep is more readily induced, and irritability of the spinal cord and nerves is decidedly subdued. The use of sedatives and narcotics is thus happily superseded. The brace is made by Geo. C. Frye, Surgical Mechanician, Congress Street, Portland.

The local effect of certain inhalations in bronchitic asthma is often good. In cases of great tightness and distress at the chest, a warm, steaming inhalation from Dr. Lee's steam-draft inhaler is usually soothing, especially if the larynx be very irritable. The steam of the water may be medicated by the addition of from 5 to 10 drops of creosote, or 30 drops of tincture of benzoin, with 10 of spirit of camphor. The steam-draft inhaler has the great advantage that the temperature of the inhalation can be readily modified by opening or closing the aperture for the air draft.

In bronchitic asthma I know of no better spray inhalation than that of ipecacuanha wine, recommended first by Dr. Sydney Ringer. The wine should be diluted with from 1 to 2 parts of distilled water, and filtered, that it may work freely in the atomizer. The patient must then inhale the spray, and the management should be such that the blast from the atomizer corresponds with each inspiratory act on the part of the patient. At the first sitting he should not take more than 20 doses or inspirations of the spray. The usual effect of this treatment is greatly to relieve dyspnœa, and mitigate cough and expectoration. The unusual effects are sudden and marked increase of dyspnœa in a few individuals who have a peculiar susceptibility to the action of ipecacuanha; and at times I have known vomiting occur some short time after the use of the spray, in consequence of the wine collecting in the mouth and being swallowed by the patient. This contingency is easily avoided by making the patient spit the collected wine out of his mouth.

The antiseptic inhaler invented by Dr. Sinclair Coghill, of Ventnor, so invaluable in many cases of pulmonary consumption, is often a great comfort to those who have bronchitic asthma. This instrument, as made by Maw, Son, and Thompson, is a plated cup perforated for the admission of air, and containing within it a second perforated plate, while between the two is interposed a thin layer of wool on which a few drops

of the inhalant are sprinkled. (One of the most generally useful inhalants is this:

℞ Creosoti,
Tinct. iodi,
Ætheris, ... āā ʒ i.
Spir. vin. rect., ... ad f. ℥ i.
M. Drops for use in inhaling respirator.)

The apparatus thus prepared is secured by an elastic band over the patient's mouth, and it has proved a great boon to many of my hospital and other patients, who have had to encounter the cold morning air.

CHAPTER IV.

REMEDIES FOR ASTHMA.

THE following clinical lecture, delivered by me at the City of London Hospital for Diseases of the Chest in July, 1884, contains some of my most recent experience in the treatment of asthma; and although some things are repeated that have been already mentioned, I have judged it best to give the lecture in full:

As a matter of observation and experience, I find that persons, from the high to the low, are disposed to regard asthma as a complaint to be endured rather than cured. In support of this statement I can bring forward the sayings of paupers in the workhouse, and of more favored people enjoying time-honored titles and living in the best parts of London. Reflect, however: Are we worse off in our chances of curing asthma than we are in respect of many other chronic diseases? Is epilepsy often cured? Then again, gout. "Strange thing," people sometimes say, "but the doctors can find no cure for gout." Pulmonary consumption is not so curable as we could wish, and there are, to my present knowledge, cases of skin disease that baffle all the best therapeutic talent in London and many other large towns. The curative action of drugs is more critically tested in chronic than in acute disease. Diseases of an acute type, such as pneumonia, measles, etc., usually tend to a natural recovery, while with chronic disease the tendency is just the other way. Chronic disease slowly grows, as it were, into the very constitution of the patient, so that the disease appears to become a part of his very being, and he gets to regard it as his companion for life; accepting gratefully such remissions as circumstances or the art of the doctor may from time to time obtain for him.

Spasmodic asthma, though by no means a dangerous disorder, is cruel in the way it deals with its victims. Asthmatics are usually persons of ability and strong nervous energy, and

often in the midst of daily and successful work are seized upon by the asthma, and after a hard day's work the sufferer has to pass a night sitting up in bed, gasping for breath, and inhaling all manner of smoke and vapor in order to obtain ease for his respiration. In the morning generally the spasm remits, and then comes the routine of daily work, after the preparation of such a night as that I have portrayed.

Therapeutic experiences in asthma can be obtained in an interesting form from the life-histories of some of the physicians of years long passed away. Floyer, who wrote on asthma in 1717, and who seems to me to have known almost as much of the mechanism and nature of the complaint as we do now, was a great sufferer. His medicines were chiefly of the evacuant class; of anti-spasmodics he does not appear to have had a very high opinion. General Gent, on the other hand, who, in 1802, introduced the use of stramonium smoke as a means of relief for his asthma, believed in its anti-spasmodic power so firmly, and resorted to it so freely, that he is said to have fallen a victim to the excessive use of his favorite remedy.

An intimate knowledge of pathology does not appear necessary or essential to guide us to the selection of a remedy for asthma. When we review the large number of remedies that find and maintain a good repute with the public for the relief of the asthmatic fit, such as Himrod's powder, Joy's cigarettes, the ozone-paper of Huggins, and the like, we do not find the inventors of these much-prized remedies to have been men distinguished in pathological research. Nevertheless, it will help us in choosing a medicine to bear in mind that asthma is a spasm of the bronchial muscles which surround the smaller air-tubes, with more or less congestion of the bronchial mucous membrane. The expiratory character of the dyspnœa in the case of old asthmatics with rounded chests should also be borne in mind. Very often such a patient will volunteer the statement that the difficulty is to get the air out of, not into, the chest. Sometimes we see spasmodic asthma coming distinctly as a secondary affection upon bronchitis. This may be called bronchitic asthma. I briefly indicate these distinctions inasmuch as they bear on the matter of remedies for asthma, with which our present purpose lies. The climatic treatment of spasmodic asthma can be soon disposed of. In the purely

spasmodic form of the complaint, where the patient goes to bed in excellent health and then is taken about three or four in the morning with sudden constriction of the chest, so that he has to sit up with his head bent down on his knees and gasp for breath, the best advice to give is to tell him when he comes to a place in which he finds he can sleep peacefully all night to stay there, for it is notorious that the finding of a fit climate for these cases is a matter of pure experiment. Remarkable tales are told of the vagaries of asthma in seizing severely on one asthmatic on the very same spot where another is rejoicing in having escaped from his harassing enemy. Dr. Birkett relates the curious case of two asthmatics, one of whom could only breathe in London, the other could only breathe at Norwood. If they attempted to go, the one to Norwood, the other to London, they were stopped on the journey by asthma, and very curiously they were both stopped at the same spot, which was Camberwell Green. Individuals whose asthmatic seizures are caused by, or essentially associated with, irritative bronchitis, have their line of climatic treatment more definitely marked out; for I believe I am correct in saying that these cases of bronchitic asthma will do well, and find their lives lengthened and more enjoyable, in a place possessing a mild and equable climate, and an air that is not dry and exciting. Torquay and Bournemouth, Ventnor and Hastings are good resorts; especially during the autumn and spring months. I have heard of the case of a patient who was attacked by a severe bronchitis with much spasm during a bad London fog. He got no relief till he went to St. Leonards. While there he coughed up much dark, sooty-colored expectoration, and recovery went on rapidly. The soothing air of St. Leonards relieved the spasmodic state of the bronchial muscles; they were enabled to act rhythmically, and so to clear the lungs of all the foulness of the London air that was clogging and oppressing them.

When we seek to relieve the urgent dyspnœa of asthma by inhalations, we generally use substances that have been found to act locally as relaxors of spasm. Chloroform cautiously employed is pre-eminently useful in giving prompt relief to the asthmatic fit. It is superior to, and less dangerous than, the nitrite of amyl; but the danger is that the patient becomes too fond of the chloroform, for the way in which I have known

chloroform consumed by asthmatic men and women has more than once caused me real alarm and apprehension. If nitrite of amyl be employed, 2 or 3 drops should be inhaled from lint, and if the breathing be noted to become slower and deeper, it will be a sign that the inhalation is likely to relieve before long.

Iodide of ethyl, or iodic ether, was introduced as a remedy for asthma in 1870 by M. Huette. Our experience of its use in spasmodic asthma at this hospital has been favorable. Six or eight drops of the iodic ether may be inhaled from a piece of lint held on the palm of the hand. A former clinical assistant, Mr. Macdonald, was able to detect the presence of iodine in the expectoration, and also in the urine of those who had inhaled the iodic ether. In the dyspnœa met with in fibroid phthisis, and in old-standing bronchitis, the iodic ether certainly is beneficial.

In the case of Annie E., æt. sixteen, from Tunbridge Wells, we found the sudden attacks of asthma to be decidedly relieved, and the frequency of their recurrence diminished, by the inhalation of 10 drops of iodic ether as soon as the breath difficulty commenced. The cough was also relieved, and expectoration facilitated. The note speaks of nitrate of pilocarpine, gr. $\frac{1}{24}$, lobelia, and citrate of caffeine, given internally, not appearing to afford an amount of relief equal to that obtained from the iodide of ethyl.

Burning nitre-paper fumes are so well known as a time-honored remedy for the asthmatic fit, that I need say but little on the subject. The more sudden and spasmodic the attack, the greater is the chance of relief from the nitrous fumes; and these must be furnished abundantly till the atmosphere becomes unbearable to a person whose lungs are healthy, for then it is that the asthmatic sucks in the medicated air with comfort and relief. Analysis of the nitrous vapor has proved in it the presence of cyanogen, nitrogen, carbonic acid, and ammonia; whether the specific action of the paper when burnt is due to one of these bodies more than another I cannot say.

The nitre-paper has been medicated in various ways, and the addition of iodide of potassium, as in the ozone-paper of Huggins, may be an advantage. In cases of bronchitic asthma, where there is some amount of actual bronchitis present in the air-tubes, burning nitre-paper often aggravates the

distress of the patient. It is in these cases where iodic ether answers much better than the nitrous fume, and where preparations of arsenious acid or phosphorus internally are very effectual remedies. Of anti-spasmodic powders and cigarettes for inhalation there is an endless variety known to most of those present. Slade's stramonium cigarettes, and Savory and Moore's datura tatula cigarettes and powder, answer well in cases where there is much spasm and great inspiratory dyspnœa, as if the lung was closed and wanted opening. Belladonna and lobelia powders are also valuable anti-spasmodics. The celebrated Himrod's powder, so much used for asthma, does not seem to contain stramonium; probably it contains belladonna. The objection to these fuming powders and cigarettes is the dryness of throat and headache which so commonly follow their employment.

Of internal remedies I am bound to speak well of caffeine, usually given in the form of what is known as citrate of caffeine, though chemists say there is no definite combination between the citric acid and the caffeine. Dr. Fowler, of Wakefield, first drew my attention to this citrate of caffeine in 1878, on account of its marvellously curative action in his own case after the failure of an immense number of remedies. The citrate is best given in doses of 1 to 5 grains dissolved in warm coffee, and it very seldom fails to give relief to the asthmatic paroxysm. We want more investigation as to the mode of action of caffeine. M. Leblond says it regulates the heart, augments its force, promotes diuresis, and is safer and more certain in action than digitalis. In poisonous dose it is said to paralyze the medulla. While I find much said in English and French works on the value of caffeine in cardiac dropsy, I do not find anywhere mention made of its great power in relieving asthma. I have heard (Medical Society's Proceedings, Vol. VI., 405) of as much as 60 grains of citrate of caffeine, taken by mistake, producing muscular tremors, vomiting, and rather alarming symptoms, which were relieved by digitalis. The only case in which I have actually seen serious symptoms follow on the use of caffeine was in the case of a young medical man, who had severe attacks of dyspnœa, resembling asthma. To him I gave 1 grain of citrate of caffeine: the effect was at once to relieve his breathing, and he was rejoiced at the speedy action of the medicine: but soon

there came on a most deadly faintness from which he was with difficulty restored. At the time one could not help thinking the caffeine might have been the cause of this faintness, but more extended observation of this case showed me that without warning very alarming fits of syncope at times attacked this patient, and I heard that he eventually died in one of these seizures. The case was one of those that now and then come under notice as cases of asthma, but which are really indications of profound nervous lesions. Curious to say, three cases of nervous dyspnœa that have thus come under my notice have all been in the cases of medical men. Two died and one is yet alive, but hemiplegic on his right side.

The second edition of Dr. Hyde Salter's work on asthma was published in 1868, and I have vainly looked in it for mention of arsenious acid as a remedy for asthma. In 1865, among out-patients at this hospital, I accomplished some striking and durable cures of bronchitic asthma by means of arsenic in the form of 2 or 3 minims of Fowler's solution. Arsenical preparations give vigor and support to the respiration, and enable people to ascend mountains without confessing the toil by a single sigh, hence one judges that in many forms of asthma arsenical medicines may prove advantageous.

One case taken from several will give the indications for arsenic:

February 12th, 1883.—C. W., æt. fourteen, has been asthmatic from the age of two years. He coughs violently, and when the asthma takes him his lower chest is drawn in. Tongue large, throat congested. Pulse 88. Respiration harsh and loud. Heart normal. At Kimberley, near the Orange River, he was quite free from asthma. He was ordered twice daily 2 minims of Fowler's solution, with 2 grains of iodide of potassium in water. On March 19th, I heard that he had not required any medicine for the last fourteen days, and seemed perfectly cured.

The liquor sodæ arseniatis is a preparation that may be given with much hopefulness in bronchial and bronchitic asthma. I have never seen any unpleasant effects follow on the employment of arsenic; but when there is hæmoptysis and weakness of the heart I have found it fail to give relief.

I wish time would allow me to enlarge upon the effects of arsenic-eating, for they are curious. At Salzburg Arsenic Works the men who stand the fumes best eat arsenic daily with their food. A gentleman who came to learn assaying at the age of seventeen years was advised to eat a bit of arsenious acid every day to enable him to bear the exposure to the vapor. He continued the practice up to the age of fifty. "Twice," said he, "I tried to give up my arsenic, but on each occasion I experienced faintness, sweating, loss of sleep, and violent palpitation of the heart. Inflammation of the lungs followed; I was laid up for nine weeks, and should have died had I not returned to my arsenic." Arsenic-smoking in a pipe is known as the Chinese remedy for asthma. Cauvin ("Lancet," 1861) reports the case of an asthmatic lady, who, in an experience of twenty-five years, found no remedy equal to $\frac{1}{4}$ grain of arsenious acid three or four times a day, mixed in a stramonium cigarette.

I pass over many remedies for asthma, well known to most, to mention a new one that we have recently been employing—the euphorbia pilulifera, from Australia. A decoction of the dried plant is made in the proportion of $\frac{1}{2}$ oz. to 1 pint of water, and when cool 100 mm. of spirit of chloroform are added. Of this decoction the patient takes a wineglassful three times a day. When it does not agree you may expect depression and faintness to show themselves. In bronchitic asthma, with emphysematous lungs, the euphorbia seems of some service. I have notes of four cases in which we have tried it. In the case of Georgina M., æt. twenty, living in London, and for three months an in-patient at the Middlesex Hospital, on account of extremely severe paroxysms of asthma, with tendency to congestion of lungs, we found, for a time, the euphorbia of service, while caffeine, belladonna, and an immense number of other medicines, including iodide of potassium in 15-grain doses, had no curative effect whatever. Emily K., æt. thirty-two, from a marshy part of Essex, was twice in Victoria Park Hospital for bronchitic asthma, with occasional paroxysms of severe dyspnœa, when she turns livid in the face. The euphorbia certainly answered well here. It kept off the asthma, while the cough and muco-purulent expectoration diminished under its employment. It did more good than any other medicine, and she left the hospital on December 29th, 1883, very much relieved.

James B., æt. forty, with bronchitic asthma and tendency to congestion of lungs, was admitted October, 1883. He said he had taken every remedy that he had seen advertised for the cure of asthma. Euphorbia suited him and gave relief; but did not seem equal to a combination of iodide of potassium and carbonate of ammonium. His asthma and bronchitis were of nine years' duration, due to cold taken in his employment as a coachman.

There are cases, met with in pessons advanced in years, where the chest is round and barrel-shaped; it is extra-resonant on percussion, and this resonance extends very low down in the chest, giving the impression that the diaphragm does not move up and down as it ought to do. The diaphragm is mechanically presed downward by the over-distended lungs, and cannot rise to attain its proper position in expiration. A line or zone of congested capillary vessels can sometimes be traced on the skin along the line of attachment of the diaphragm. It is in very confirmed cases that I have noticed this sign present.

On auscultation we scarcely hear any inspiratory sound. The lungs are so distended that but little air can be taken in with inspiration. With this stagnant condition of respiration the patient complains much of coldness in his extremities, his lips and nose look dark and congested, and his general condition is one of dulness and apathy. At intervals the bronchial muscle is thrown into a state of rigid spasm, and thus attacks of asthma come on, and greatly distress the patient. Ordinary expectorants and sedatives are of very little use as remedies in this form of asthma, but I have seen much good done by liquor strychniæ or pills of extract of nux vomica. The liquor may be given in doses of 3 to 5 drops, with dilute phosphoric or hydrochloric acid. In cases where expiration is much prolonged, with collection of phlegm in the lungs, I have found both strychnia and nux vomica useful.

In the case of a patient lately under my care with barrel-shaped, almost circular, chest and frequent asthmatic seizures, we were able, in consultation with Dr. Cayley, to try the effect of the inspiration of compressed air from Waldenberg's spirometer. The benefit was slight, and the trial of expiration into rarefied air of no benefit whatever. Subsequently I learned that the inspiration of oxygen gas had proved useful;

but what did more good than anything else was going through a course of severe manipulation at the hands of a celebrated Swedish practitioner. The abdominal muscles were well rubbed and shampooed, and thus their action as muscles of expiration was roused and stimulated. The diaphragm also was powerfully brought into play, with the result of curing the asthmatic seizures and bringing improved appetite and increased vigor to the circulation, so that the patient felt warm and cheerful.

Of the treatment of hay asthma and summer catarrh I have not much to say. The pollen of grasses and of flowers appears to be the cause of this complaint, and various respirators have been invented to protect the nostrils against the invasion of the pollen. A snuff, made by rubbing well together 20 or 30 grains of iodide of sulphur and 200 grains of powdered liquorice-root, has seemed to me decidedly of service in some cases. The powder must be made as fine and impalpable as possible, and then a little of it may be snuffed into the nostrils. Gargling the throat and bathing the nostrils and eyes with a very weak solution of potassium permanganate in water is often very comforting. It is well to commence with 5 drops of the B. P. solution of the permanganate in a tumblerful of water, and the strength can be increased by degrees. The inhalation of very dilute chlorine gas, or of chloride of ammonium from one of Burrough's inhalers, is an efficient mode of obtaining temporary relief.

Of internal remedies I believe belladonna to be the best when the defluxion from the mucous surfaces is very profuse and distressing. From 3 to 6 mgm. of the succus belladonnæ should be taken in water every four hours, and such experience as I have had of this remedy has been certainly encouraging.

CHAPTER V.

ASTHMA IN RELATION TO GOUT, PHARYNGEAL CATARRH, AND RHEUMATOID ARTHRITIS.

THE remarkable way in which gout may manifest itself, sometimes in the form of great nervous perturbations in the system, at other times of sudden local congestions, makes it easy to understand the intimate relation that there is between asthma and gout. Gout may disturb the circulation by its irritant action on the nerves which control the blood-vessels and circulation. Thus is caused that pulsation of the abdominal aorta that often alarms the gouty dyspeptic, and makes him fear that he has got abdominal aneurism.

Dr. Matthew Baillie has recorded the case of a gentleman who suffered palpitation of the heart for six months without obtaining any relief from medicine; but a fit of the gout coming on in his foot, the palpitation suddenly and entirely left him.

Scudamore says, in his treatise on gout, "I have seen sympathetic affection of the diaphragm, in one case, amount to so great a degree of spasmodic asthma, attacking the patient at first twice and afterward once in the twenty-four hours, so that each paroxysm was dreaded with horror from fear of suffocation. These attacks lasted three hours, and certainly and speedily followed any error in the quantity or quality of the dinner meal."

The way in which gout in the bood may induce dangerous fulness and congestion in the vessels is shown by the history of those who have tried to cure acute gout in the foot by means of cold applications. Alarming and sometimes fatal congestive apoplexy has been the result.

It is worth while to observe the way in which the intestinal canal is often disturbed in gouty people. Piles, every one knows, are common symptoms of gout. These piles are mainly caused by irregular action in the muscular coat of the

bowel, or irregular peristalsis leading to obstruction and pressure on the vessels. This irregular action is shown in various ways. Sometimes the muscular fibres of the large intestine are torpid and will not contract, so that the bowel becomes unduly loaded with fæcal matters. At other times there is very irregular and spasmodic action, so that a kind of dysenteric condition is established with intestinal catarrh, and much pouring out of mucous. Irritating matters in the bowel of acrid and acid nature, such as are generated in the intestines of gouty people, cause this irregular action, and the habitual and too frequent use of purgatives most certainly aggravates, as well as causes, this evil condition. The scanty watery evacuations often make the patient think himself the subject of stricture of the bowel.

The muscular coat of the bowel gets into very much the same condition that the bronchial gets into in cases of catarrhal asthma. In each case the mucous membrane is irritated, and the circular muscular fibres lying beneath the mucous membrane are also irritated by contiguity of tissue, and are either thrown into spasmodic action or else are paralyzed and refuse any action. Inflammation round an intestinal ulcer has been known to cause all the symptoms of intestinal stoppage by paralyzing the muscular coat of the bowel. Expectorant medicines fail to relieve the bronchial spasm and irritation, and purgatives notoriously fail in bringing about healthy action in the bowels. Suitable diet and regimen, and the judicious employment of such anti-spasmodics as belladonna and sometimes opium, seldom fail to give more or less relief to both bowels and bronchi. I have seen opium and belladonna act as admirable laxatives in cases where the bowel had regularly struck work under the influence of strong cathartics, responding merely by scanty watery evacuations. The soothing effect of a proper climate in promoting rhythmical action of the bronchial muscle is exemplified by the case noted at p. 708.

Chronic pharyngeal catarrh, with mucus about throat and fauces, and at times buzzing noises in the ears, from catarrhal obstrucion of the Eustachian tubes, are common symptoms in gouty asthma and bronchitis.

In 1881, I had under observation a case of gouty asthma. A wine merchant, thirty-eight years of age, observed that

his attacks of asthma always came on if he had any disorder of stomach. He had a cough, with scanty, glutinous, difficult expectoration, and had coughed up streaks of blood on rare occasions. Liable to pains about joints, and at times has much abdominal pain across the colon. The motions often contain much white mucus.

Urine specific gravity 1014, acid; trace of albumin. Pulse 88; heart's sound feeble. Tongue large, red, and clean. Appetite good. Chest resonant; a few mucous râles audible.

For three years I had this patient under observation; at times the arthritic pains were very severe, at other times the asthmatic attacks were especially distressing. On the whole he did fairly well, for he gained flesh, but no remedy cleared away the asthma and joint-pains. The medicine that was superior to most others was arseniate of soda in small doses.

The experience of this and several similar cases has convinced me that the great thing is to diet the patient carefully. In very obstinate cases of bronchial asthma, alternating with gout, it is a good thing to make the patient abstain entirely from all butcher's meat. Let him have fish, poultry, and game, and if then there be only partial improvement let him try vegetarianism for a time.

This process of cure is trying, and but few will put up with it. Those who have tried it usually benefit in a surprising degree, and appear to take a new start in life. The gouty asthmatic has often a clean tongue and a hearty appetite; all may go well till the food leaves the stomach and gets into the intestines, then his troubles begin. Restlessness, heat, and itching of skin, with frequent desire to pass water, and perhaps diarrhœa, are all signs that nutrition is out of order. Acid eructations, tasting of food taken many hours previously, and heartburn will show that the stomach digestion is imperfect. By prolonging the intervals between meals, reducing the quantity of food taken, and simplifying its quality, much good may be done. If the tongue be angry-looking and red, then light farinaceous food and very sparing allowance of meat will be the general line of diet to be followed. When the tongue is large, flabby, and indented at the edges, care must be taken not to oppress the stomach by a mass of food, but to give small amount of nutritious aliments; and here meat in small quantity will probably be found more suitable than

heavy meals of farinaceous food. Overfeeding is the bane of these cases.

Much drink should not be taken with the food, but after the meal digestion is promoted by a small quantity of plain or aërated water sipped at intervals. Undue acidity of food is mitigated, and healthy movement of muscular coat of stomach promoted, by the gentle stimulus of the cold water. If heartburn and acidity are severe, then a tumblerful of hot water appears to me a first-rate cure; and if this be taken pure and simple, without adding soda or magnesia, it will not lose its powers of cure by repetition. Aërated waters, taken with the food, distend the stomach, oppress the breathing, and become fertile causes of flatulent dyspepsia and weak stomach; and plain water taken in immoderate quantity with sweet food is very likely to set up acetous fermentation in the stomach.

One glass, and never more than two, of good Manzanilla sherry, or half a wineglassful of whiskey or brandy with water, but on no account sugar, will agree well with many persons at their dinner, and will prevent their feeling an undue degree of thirst. Some are suited best by claret, but on no account should two kinds of alcohol be taken, and at times a fast for a week or so from all forms of alcohol is beneficial. Malt liquors and all sweet or effervescing wines should be entirely avoided.

Total abstinence as a permanency suits some of the gouty asthmatics, but I expect Sydenham was quite right when he said: "If you drink wine you will have the gout, and if you do not drink wine the gout will have you." The drinking of any form of alcoholic drink on an empty stomach is a practice pretty sure to rivet firmly on the patient gout, catarrhal asthma, and all manner of ills.

Asthma being so very often kept up by the persistence of pharyngeal and nasal catarrh, a complaint usually of gastric origin, makes it well to insist on these matters of diet. Very hot drinks are causes of throat congestion, hence tea and coffee must be taken moderately cool. Gargling the throat with a weak solution of borax in warm water (60 grains in half a pint), I find a method of some value in the treatment of this pharyngeal catarrh. In using the gargle it is necessary to throw the head well back, or even to lie flat on the back, so that the posterior part of the throat may be well

medicated. Frequent topical applications by means of brushes appear to me to do more harm than good; but if the tonsils are large, then pencilling them over with a solution of iodine in glycerin (3 grains to 1 oz.) is very useful, and preferable to the employment of strong astringents.

As internal remedies to overcome this chronic pharyngitis the liquor sodii arseniatis in 2-minim doses, taken in plain water or in Vichy water, is worth much. Often the association of the iodide of sodium in dose of 2 or 3 grains adds to the curative action of the arseniate. Sulphur I have tried in various forms, such as the bisulphide of carbon in dose of 1 drop, and the potassa sulphurata in dose of $\frac{1}{4}$ to 1 grain; but these medicines, though of service often in relieving the asthma, do not appear to me to have much curative action over the pharyngeal irritation.

I have often been impressed by observing the wonderful relief that is obtained in many cases of gouty bronchitis and asthma by judicious depletion of the abdominal viscera by a course of mineral waters, such as those of Carlsbad. What is known as abdominal plethora and congestion of liver powerfully impede the free descent of the diaphragm, and patients who are thus affected very soon get out of breath with the smallest effort. The heart palpitates, and this general disturbance is sometimes put down to weakness, and treated by full diet and free stimulation. I have known cases where meat has been taken regularly two, if not three, times in the day; while at bedtime the day has been finished with a strong glass of either whiskey or brandy with hot water and sugar. The result has been an almost alarming condition of breathlessness, cough, and heart difficulty. Reducing the allowance of meat and withholding stimulants, while moderate doses of Carlsbad salt are taken in hot water first thing in the morning, are means which seldom fail to give more or less relief, and not uncommonly that change for the better is really something marvellous in the eyes of the patient and friends.

In some of these cases where there is evidence of pulmonary emphysema and dilated heart, judgment is needed so that we do not go to work with anything like violent purgation, a thing to be strongly deprecated. Another practical point is to take care that the asthmatic who has emphysema and dilated heart be not allowed to go to any high altitude for

change of air. The high Alpine regions, so beneficial in cases of threatened consumption, are positively dangerous for one who has much emphysema of the lungs with dilated heart. Condensed, not rarefied, air is what these old asthmatics require to make them comfortable.

The relations of asthma to rheumatoid arthritis, sometimes called rheumatic gout, are not very clear. I have known a genuine asthmatic get rid of the asthma, and then find himself gradually invaded in his joints by stiffness, cracklings on movement, and some thickening. The fact is interesting if, as Dr. Buzzard has suggested, rheumatoid arthritis is due to disorder or disease of certain centres of nutrition in the medulla oblongata. Furthermore, I have often been struck by seeing the beneficial effect of small doses of arseniate of sodium in relieving the joint symptoms in rheumatoid arthritis; and that this same medicine is one of very great value in curing persistent asthma is now a recognized fact in therapeutics.

Seeing how very often, in gouty people more especially, the inflamed and irritable condition of the throat and pharynx keeps up the tendency to bronchial spasm in the lungs, it is well to caution such patients against the too frequent use of the various fume inhalations by powders and cigarettes. These means are undoubtedly good as palliatives, but their frequent use tends to heat and dry the throat, and so add much to the permanent discomfort of the gouty asthmatic.

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CONVULSIVE SEIZURES.

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CONVULSIVE SEIZURES.

CHAPTER I.

As preliminary to my address, I have to mention my great obligations to Dr. Ferrier. There are very few men of the day by whom the art and science of medicine have been so greatly helped. It is only since his remarkable researches that medical men in this country have studied convulsions in a thoroughly realistic way. Without further particular acknowledgments, I make the general one—that all over the field of neurology I am profoundly indebted to Dr. Ferrier.

Convulsions and other paroxysms are owing to (1) sudden, (2) excessive, and (3) temporary nervous discharges. (A term introduced by Dr. Edward Liveing in his masterly work "On Megrim" is "nerve-storm." This term has met with much favor, and the conception it stands for has been of great value in elucidating some very complex problems in neurology.) There are nervous discharges in all the operations of health. I should use the term "explosive discharge" for the abnormal liberation of energy in convulsions, were it not that physiologists speak of "explosive decomposition of the muscle's substance" which gives rise to contraction of the muscles in health; and thus it may be that normal nervous discharges, those in the operations of healthy people, are "explosive." So then I use the word "excessive" for the discharges which have the three characters mentioned, the words "sudden" and "temporary," or the word "paroxysmal," being understood; when dealing with convulsions, it will not be always necessary to add the

term "excessive." I shall, however, occasionally use "explosive" for states of cells of a discharging lesion, qualifying it by the word "highly," and sometimes the term "high instability." I do not speak of "disorderly discharges;" for if in some ways the expression be correct, it hides from us the fact that the most brutal-looking convulsion is only the sign of a departure by a vast excess, and by a caricature from normal nervous discharges.

I have always assumed, and shall continue to suppose, that convulsion results from excessive discharges of nerve cells, meaning of course liberation of energy during rapid decomposition (katabolism) of some matter in, or of part of, those cells. I shall frequently speak of cells concerned with excessive (primary) discharges as constituting a "discharging lesion," and sometimes of them as making up a "physiological fulminate," or occasionally, using Mr. Horsley's term, of their being together an epileptogenous focus." Some material of the cells which make up the discharging lesion has, by morbid nutrition, become of very high tension and of most unstable equilibrium (briefly of high instability), and occasionally discharges excessively. I do not assert that excessive (primary) discharges producing convulsions always depend on a persistent state of high instability of cells; to give but one example to the contrary, excessive discharges beginning in a healthy rabbit's respiratory centres are induced by rapidly bleeding the animal to death, or by quickly asphyxiating it.

The discharging lesion, though I speak of it as persistent, is yet varying. (The rapid reader must not take "persistent discharging lesion" to be "persistently discharging lesion.") The discharges are occasional. After their excessive discharge the cells are, no doubt, far below the degree of stability (properly comparatively slight instability) of normal cells; they will re-attain a highly abnormal degree of instability—again become highly explosive—by further morbid nutrition. These qualifying remarks should be borne in mind, and especially when I come to speak of the discharging lesion as a fulminate—it is occasionally fulminant.

I make three classes or kinds of convulsions. Convulsions, or I will say fits, differ in kind, according as centres discharged differ in rank or, speaking more definitely, as the centres first

engaged in paroxysms make up different evolutionary levels of the central nervous system. I have several times suggested that there are three levels of the cerebral (central) nervous system; each is sensori-motor, and each represents impressions and movements of all parts of the body. I speak briefly of what I suppose to be the hierarchy of the centres of the nervous system as a basis for the classification of fits.

1. The lowest or first level is roughly and incompletely defined as consisting of cord, medulla, and pons, and more completely, and yet still roughly, as being that sensori-motor division of the central nervous system to and from which pass nerves (all cranial and all spinal nerves) for every part of the body. This level, speaking of its motor elements, represents simplest movements of all parts of the body by a series of lowest motor centres (lowest motor centre being a proper name for a centre of the lowest level), from those in the floor of the aqueduct of Sylvius, for simplest movements of the ocular muscles, to those of the sacral cord, for simplest movements of the muscles of the perineum. This universally representing level is cerebro-cerebellar; it is at once the lowest level of the cerebral system and of the cerebellar system. For the present I ignore the higher levels of the cerebellar system, and go on to speak of two higher levels of the cerebral system. These levels are, as the lowest level is, sensori-motor, but I find it possible to illustrate by motor centres only, not, however, believing that these so-called "motor centres" are purely motor. 2. The middle or second level (its motor province) of the cerebral system is composed of centres of the Rolandic region (so-called "motor region" of the cerebral cortex), and possibly of the ganglia of the corpus striatum also. It represents complex movements of all parts of the body from eyes to perineum (re-represents). 3. The highest or third level (its motor province) of the cerebral system is made up of centres of the præ-frontal lobes (highest motor centres, motor division of the "organ of mind"). It represents most complex movements of all parts of the body from eyes to perineum (re-re-represents).

There are, I submit, three kinds of fits corresponding to the three evolutionary levels. It is convenient to speak of the three kinds in an order different from that used in stating the

levels. (2) I mention epileptiform seizures first because their localization is not doubtful. (They were first described by Bravais in 1824.) They are "middle-level fits"—that is, they are produced by excessive discharges beginning in parts of the middle level (motor province) of the cerebral system ("motor region"). My hypothesis is that (3) fits of epilepsy proper ("genuine epilepsy" of some nosologists) are "highest-level fits," and that many of them, not all, are produced by excessive discharges beginning in parts of the præ-frontal lobes, highest level (motor province) of the cerebral system. Although the præ-frontal lobes are not experimentally excitable, I suppose it will not be denied that their cells katabolize and liberate energy in their normal activities, and it is not unreasonable to suppose that parts of them may, by pathological changes, become highly explosive, so that they occasionally discharge excessively. So that if the præ-frontal lobes are divisions of the "intellectual centres," as no doubt they are, and not, as I think, motor too, excessive discharge beginning in parts of them may produce epileptic fits. Of course this is speculative. I am not aware that any one pretends to know the seat or the pathology of cases of "genuine epilepsy." I do not use the term "cortical epilepsy," because both epileptic and epileptiform seizures are, to my thinking, cortical fits. (The difference in meaning assigned to the two terms epileptic and epileptiform must never be lost sight of.) (1) I think that there are "lowest-level fits;" these are fits produced by excessive discharges beginning in parts of the lowest level, a level which is common to the cerebral and the cerebellar systems. I suppose that most of them are owing to excessive discharges beginning in centres of the bulbar and pontal regions of the level; hence, I sometimes use the term "ponto-bulbar fits." With regard to epileptic form of epileptic fits, I deal almost exclusively with cases of patients subject to fits—with, so to speak, "chronic cases."

I have so far spoken only of three kinds of fits. No doubt there are varieties of each kind. While the kind answers to the level, the variety of each kind answers to the particular part of the level in which the excessive discharge begins. It is certain that there are varieties of (2) epileptiform seizures; each is marked by a particular place of onset of the convul-

sion. There must be at least as many varieties of (3) epilepsy proper as there are different "warnings" of the paroxysms. Presumably there are varieties of (1) ponto-bulbar fits.

Then, of course, there are degrees of each variety dependent directly on degree of the primary discharge and indirectly on that of the secondary discharges. (2) For example, there are degrees of that variety of epileptiform seizures marked by the first spasm being of the thumb. There are many ranges of it, from convulsion almost limited to a thumb, thence onward to universal convulsion. (3) Everybody recognizes that epileptic attacks occur in two vastly different degrees, *les petits maux* and *les grands maux*, and there are sub-degrees of each of these degrees. (1) There are degrees of convulsion in lowest-level fits produced by Brown-Séquard's method in guinea-pigs. There are, no doubt, degrees of respiratory fits produced in certain lower animals by rapid bleeding, by ligature of the great arteries of the neck, and by sudden stoppage of respiration.

To repeat, "fit" is a term used to include convulsive paroxysms of all kinds dependent on excessive discharges beginning in any part of any one of the three levels, the epileptic and epileptiform beginning in one half of a level. There are three kinds of fits—(1) ponto-bulbar, (2) epileptiform, and (3) epileptic. There are, the supposition is, varieties of each kind and degrees of each variety of each kind.

As I shall not have time to deal with lowest-level fits, will merely mention what I think are some fits of this kind, not classifying them, but making a rough arrangement into three groups. (1) Respiratory fits (respiratorily beginning from primary discharge of the head respiratory centre). I think ("Brain," April, 1886) that fits of laryngismus stridulus come in this category, but Dr. Semon thinks they are cortical seizures. Respiratory fits are easily induced in animals, and are described in all works on physiology when asphyxia is considered. Kussmaul-Tenner fits are respiratory fits. (2) Fits produced by convulsant poisons (fits from nitrous oxide and curara are respiratory fits). Fits are experimentally produced in animals by absinthe and camphor. (Many years ago Dr. George Johnson described convulsions in man from poisoning by camphor.) Possibly some fits in renal disease (some so-called uræmic fits) and, it may be, fits occasionally

part of a constitutional disturbance after urethral lesions, are lowest-level fits from home-made poisons, as those just spoken of are supposed to be from foreign poisons. (3) A condition for fits consequent on certain injuries of the cord or sciatic nerve in guinea-pigs (Brown-Séquard). These are so well known that mere mention will suffice. There are often fits attending the onset of infantile paralysis; these are very difficult of explanation. I submit the hypothesis that they are lowest-level fits produced by action on the ponto-bulbar centres of ptomaines the result of disintegration of nervous matter of the anterior horns; if so they come in group 1.

In all severe lowest-level fits it is supposed that the primary discharge of ponto-bulbar centres not only induces discharge of other lowest motor centres, but also that by intermediation of sensory ("ascending") fibres it discharges centres of higher levels. (I never thought of implication of higher centres in these or any other fits by intermediation of sensory nerves until after consideration of the researches of Victor Horsley and Binswanger.)

It is necessary now to speak of abnormal affections of consciousness with regard to kinds of fits. I presume that there is loss of it in severe fits of all kinds. Consciousness is not a function of the highest cerebral centres; it is simply concomitant with their functioning. There is no physiology of the mind any more than there is a psychology of the nervous system. On the basis of mere concomitance mental symptoms (synonymously abnormal states of consciousness) are, strictly speaking, only signs to physicians of what is not going on or of what is going on wrongly in part of a patient's material organization. Thus cessation of consciousness at, or close upon, the onset of an epileptic fit is of value to physicians as a sign that the correlative physical process, the excessive discharge, begins in some part of the "organ of mind," or, equivalently, highest centres of the cerebral system; the physical process in these and all other kinds of fits is our proper concern as medical men. Consciousness is lost late in epileptiform seizures, and in those of but little range there may not be even defect of consciousness; this agrees with the empirical evidence that the excessive discharge begins in lower (middle motor) centres; probably excessive discharges are induced (upward) in the highest sensori-motor centres by inter-

mediation of sensory fibres when consciousness begins to cease in an epileptiform seizure. Availing ourselves of abnormal affections of consciousness as signs of the state of the central nervous system, we next, so to speak, put them on one side in order to study the process in fits in a purely materialistic manner.

It will be observed that I have spoken of the excessive discharge productive of fits beginning in this or that level; further, of its beginning in some part of a level. In recapitulation, the primary discharge in all kinds of fits is of some part of but one of the levels. And now I add that in epileptic and epileptiform seizures the excessive discharge begins in some part of one half (lateral) of a level; thus, so to say, in these fits the discharging lesion is "doubly local." If the discharging lesion be, as I suppose, of but a few cells, very little of a convulsion is directly due to it. Most of the convulsion is produced by intermediation of fibres between the cells of the discharging lesion and other cells of its own level and of other levels; there are induced consecutive discharges of normal stable cells. Hence the interconnecting fibres of each level and the fibres connecting the several levels with one another, and the fibres connecting the lowest level with all parts of the body (lowest level of the whole organism), have to be considered. (I shall strain the meaning of the word fibre, making it stand for any kind of nervous pathway insuring physiological union, definite or indefinite.) Consideration of these connections is essential for clear ideas of the full process in fits, the only visible part of which is convulsion; it is especially important with regard to the way by which in epileptiform seizures a very local (a "doubly local") discharging lesion causes wide-spreading and even universalization of convulsion. Again, the study of the interconnections of the levels is a necessary preliminary to the comparison and contrast of the effects of "discharging lesions" and of "destructive lesions," an essential thing in the scientific investigation of diseases of the nervous system, as I urged in my Gulstonian lectures (1869). I speak only of connections of motor centres of the levels.

Each level being bilateral is a twin series (right and left) of centres; there are connections (commissures) between "identical" centres and between "non-identical" centres of its two halves—presumably of centres of the halves as they corre-

spond for co-operation of the parts of the body they represent in joint operations by the two sides of the body. There are also connections between the centres making up the lateral half of each level. The fibres of the two sets of connections spoken of are intrinsic fibres of the level. I speak next of extrinsic fibres — that is, of those interconnecting levels. Considering for a moment all the levels, the motor path, or, as I shall say, kinetic route, extends from the highest motor centres to the muscles, which in a certain regard, being dischargeable, are centres too. (Dr. Gowers, "Diseases of the Nervous System," Vol. I., p. 116, gives a diagram of the "motor path." He makes two segments, "cerebro-spinal" and "spino-muscular." The kinetic route is a modification of his scheme.) This route (strictly the three series of motor centres are parts of the kinetic route) is in three segments: from highest to middle centres, first segment; from middle to lowest, second segment; and from lowest to muscles, third segment. I can, however, consider in detail only the second segment of the kinetic route, that connecting the middle and lowest levels. I shall, for convenience, speak of the motor centres of the right half of the middle level. There are three sets of motor fibres, kinetic lines of the second segment, uniting all right middle motor centres to all lowest motor centres—at least, to all motor centres of the left half of the lowest level, if not, as I imagine, to those of the right also.

First set of fibres of the second segment: those which have been traced (on the Wallerian method by Charcot and others) from the right "motor region" along the right corona radiata, right internal capsule, through the right crus cerebri, right halves of pons and medulla into the (left) lateral column of the cord, as low, Sherrington has found in one case, as the origin of the coccygeal nerve-roots. These fibres are (and so are the second and third sets) extrinsic of the levels; they belong to neither, and yet they belong to both in the sense of interconnecting the two. Second set of fibres: those of the direct pyramidal tract. They have been said to be traceable no lower than the mid-dorsal region. Tooth has traced them in one case as low at least as the second lumbar; these are fibres of the inner part of the right anterior (Turck's) column. Third set of fibres: comparatively recently (1884) discovered by Pitres, and seen by Schäfer, Sherrington, Hadden, Tooth,

France, and others. These fibres have been traced into the right lateral column. The degeneration in the (right) lateral column in cases of (left) hemiplegia is recognized by Charcot. So we see that the interconnection of the middle and lowest motor centres is very complex.

Some years ago[1] I inferred from the then known connections of both halves of the cord (by the first and second set of fibres) that both sides of the body are represented in the right half of the brain (I still say "right" for convenience), but that, considering the degenerated fibres were of different columns (the left lateral and the right anterior), the left and right sides of the body were differently represented in the right half of the brain. (This, I have since stated, seems to me to be but an expansion and modification of the principle of Broadbent's well-known hypothesis as to the double representation of the bilaterally acting muscles.) I should have thought the discovery of the third set of fibres rendered my hypothesis more tenable. But there are serious difficulties. The second set of fibres (those found in the right anterior column) are supposed by some great neurologists to cross to the left half of the cord.[2] The same has been said of the third set of fibres found in the right lateral column. So that it may be that all three sets of fibres pass from the right middle to the left lowest motor centres. It is, however, not really known where the second and third sets of fibres end. No wasted fibres are found in the anterior commissures. Hence, I provisionally keep to the hypothesis mentioned, and now say regarding it that the three sets of fibres may show that the right middle motor centres are connected with lowest motor centres of the left and right halves of the lowest level, and thus that the right middle motor centres represent, by intermediation of the twin lowest motor centres, movements of muscles of both sides of the body. There is still another matter to be considered before I can illustrate this hypothesis.

Having considered the whole central nervous system with the rest of the body represented by it as a sensori-motor mechanism, I now wish to urge that the motor centres of every level represent movements of muscles, not muscles in their individual character; the same muscles—that is, all the muscles—are represented in simplest, in complex, and in most complex movements by respectively the lowest, middle, and

highest motor (better "movement") centres. If so, it is possible, as I believe happens in hemiplegia, to lose one series of movements of muscles, and to retain another series of movements of the very same muscles. Thus, to take a case of left hemiplegia in which the right middle motor centres, if not destroyed, are cut off from lowest motor centres (to neglect the second and third sets of fibres), the condition is not properly described as "loss of power of the muscles" of the left arm and leg, but as loss of complex movements of the muscles of those limbs. The simplest movements of the very same muscles remain represented by the left lowest motor centres. The muscles certainly are unaffected, and when rigidity comes on the simplest movements of them are developed by overactivity of their lowest motor centres. The fixed rigid state of the left arm and leg, which clinically we speak of as a symptom, as if it were something *sui generis*, is only the algebraical sum of the co-operating and antagonizing "pulls" of the simplest movements of the muscles of these limbs.

If the hemiplegia be very slight, we may say that the muscles of the left arm and leg "are only a little weak," but the strict description is that there is then loss of but a very few complex movements of all the muscles of these limbs, with persistence of the rest of the complex and of all of the simplest movements of those same muscles. Similarly convulsion is not to be looked on as convulsion of muscles; in an epileptiform seizure the convulsion is a contention of complex and also of simplest movements. In this contention the individuality of each movement is lost. I will take a more complex case—that of a man imperfectly and permanently hemiplegic, and yet subject to occasional epileptiform convulsion of the region paralyzed; the paralysis is loss of but some of the complex movements of all the muscles of the arm and leg, and there is persistence of the whole of the simplest movements of all those muscles; when the fit comes, there is a temporary contention of the remaining complex movements and of the simplest movements of all those muscles.

The distinction between muscles and movements of muscles is exceedingly important all over the field of neurology. I think the current doctrine of "abrupt" localization would not be so much in favor if it were made. The occurrence of convulsion of a muscular region which is already imperfectly and

yet permanently paralyzed is unintelligible without that distinction. And without it we shall not understand how it can happen that there is loss of some movements of a muscular region without obvious disability in that region. This bears closely on the realistic study of the physical conditions in aphasia and insanity. We are concerned with both these morbid affections in this inquiry. The anatomical loss in cases of aphasia is of certain complex movements of the tongue, palate, etc.; there is paralysis in that sense. In cases of insanity (post-epileptic states, for example) there is, I submit, corresponding to the negative element of the psychical symptomatology paralysis in the sense of loss of some of the most complex combinations of impressions or of most complex movements, or of both.

At a first glance it seems absurd to say that there is no obvious disability in a muscular region, and also that there is loss of some movements of that region. This is, however, what I do affirm confidently. An eminent physician, referring to a previous statement of mine[3] to that effect, described my aim as being to prove two things—that a motor centre does and also that it does not represent movements. I never consciously attempted that very marvellous feat. Then, as now, I am content with the supposition that the non-disability of a muscular region when some movements of it are lost by destruction of part of a centre is accounted for by that muscular region being represented by other movements in other parts of the centre or in other centres. On this supposition of compensation we can explain prompt recovery from hemiplegia (without implicit reliance on the hypothesis of subsidence of "shock" or diminished pressure) dependent on very small destructive lesions; there is recovery when a few movements are permanently lost. That the seeming paradox holds for some cases is undeniable, as the experiments of Semon and Horsley on the cortical representation of the vocal cords show. These experiments give a crucial verification of Broadbent's well-known hypothesis for one case. Moreover, France[4] has found degeneration in the lateral column of the cord after lesions of the gyrus fornicatus in monkeys. Although in the monkeys mutilated as mentioned there is no discoverable disability in any muscular region, I submit that the degeneration France describes is proof of loss of some movements, skeletal

or visceral, or possibly of negative movements (inhibitory). It would be remarkable if there were any conspicuous disability, considering the enormous compensation given by the intact "motor region." I can now return to the question of representation of both sides of the body in each half of the brain.

I suppose that the right "motor region" represents complex movements of muscles of both sides of the body (1) as they serve in bilateral actions, (2) as they serve in alternate actions (or, I should say, with preponderance of activity on one side), and (3) as they serve in unilateral actions. But I suppose, too, that those movements which are "bilateral" are represented most nearly equally in right and left halves; that those which are "alternate" are represented less equally in the two halves (those of the left side more in the right half), and that those which are "unilateral" are represented most unequally in the two halves (those of the left side most in the right half). It is important to remember that movements are spoken of, for the so-called alternate muscles may serve in bilateral movements; they may serve with a great degree of preponderance on one side approaching unilateral movement; and the most unilateral muscles may serve in bilateral movements.

It may, however, be asked, Where is the paralysis corresponding to abrogation of the second and third sets of fibres from the right "motor region?" The existence of some weakness of the right side (the so-called non-paralyzed side) has been urged by Brown-Séquard, Pitres, Friedlander, and Gowers; but when in the difficult circumstances attending investigation of the state of the right side no disability is demonstrable, there may yet be loss of some few movements of the muscles of that side. The loss of these few movements is masked by the compensation given by the left half of the brain, which represents most numerous movements of the right side. If we take the case of excessive discharge beginning in some part of the right middle motor centres, we find that, in severe epileptiform fits, both sides of the body are convulsed. In this case compensation is "inverted"—that is to say, centres which would compensate a loss are compelled to co-operate in an excess. I shall speak further on this subject when dealing with the universalization of an epileptiform convulsion.

CHAPTER II.

I TAKE for most particular consideration the second kind of fits, epileptiform seizures (middle-level fits); they were first described by Bravais in 1824. There are varieties of epileptiform seizures. The convulsion begins in some part of one side of the body. Varieties are distinguished by the particular place of onset ("signal symptom" of Seguin) of the convulsion. The three commonest varieties of epileptiform seizures are—(1) fits starting in the hand (most often in the thumb or index finger, or in both); (2) fits starting in one side of the face (most often near the mouth, or in the tongue, or in both these parts); (3) fits starting in the foot (nearly always in the great toe).

Epileptiform fits most often begin in the animal parts of the body, and commonly in those of them which are the most animal. Thus the majority begin in the arm, and nearly all these in the most animal part of that most animal part, the hand, and most of them in the thumb or index finger, or both —the most animal parts of the whole organism. Perhaps the term animal is awkward in this connection; it is convenient in contrast to organic. We can, however, use other terms, and say that epileptiform seizures most often begin in those parts which, speaking popularly, have the most "voluntary" uses; in those parts which have great independence of movement; in those parts which have the greater number of different and more special (definite) movements, at the greater number of different intervals.

Although epileptiform seizures most often begin in the three most animal parts of the animal parts mentioned, yet as the fits go on the organic field becomes involved. In a dog poisoned by curara (respiration being artificially kept up) the animal parts are excluded, and then we see that by excessive discharges experimentally induced in its "motor region,"

effects are produced in, and are then limited to, the organic field.

There are degrees of each variety corresponding to degrees of the "severity" of the excessive discharge. Degrees are to be considered with respect to two factors—(1) amount of convulsion and (2) range of convulsion. I illustrate here quite arbitrarily by range only, and take the case of an epileptiform fit starting in the thumb. Admitting that there are many ranges, it is allowable to make arbitrary divisions (they must not be taken for real distinctions) into four ranges: (1) Terminal fit—the spasm involves, say, the hand, or some part of it only. (2) Monospasm—the arm becomes involved. (3) Hemi-spasm. (4) Bilateral convulsion—the second side being gained, the fit becomes universal. Observe the use of the word "becomes." In the range (4) there is a march of spasm from the part first seized all over the body, answering to increased spreading of the central excessive discharge beginning in some particular part of the motor region of the first half, and maybe extending to the second half, of the brain. The second side of the body (in 4) is affected later, and commonly less than the first side.

From (1) to (4) there is but a single variety of epileptiform seizure in four different degrees. Otherwise stated, a man who has had convulsion of the range (1) or (2) does not become subject to another variety of epileptiform seizure when he has also fits which, beginning in the same terminal part (hand, we are supposing), have a much more extensive range, however much more extensive it is; so to speak, that patient is subject to a fit, and has in it different degrees on different occasions. However limited or however wide the range of convulsion, the corresponding discharge begins in the very same centre of the motor region of but one, the opposite half of the brain. This means that it is the place of onset ("the signal symptom") which localizes; it points to the particular part of the "motor region" of the cells of which part the discharging lesion is made up.

I have often urged that the clinical problem in every nervous malady is of three elements: (1) Anatomical, (2) physiological, (3) pathological. In other words, we have in every case to seek (1) the seat of the lesion—the structure damaged, (2) to infer the kind of functional change of structure, and (3)

to discover the nature of the abnormal nutritive process by which the alteration of composition of nervous matter producing the abnormal functional state of structure is effected (*vide infra*).

Physiology.—Physiology deals with the dynamics of the organism—that is, with its function. I use the term function with regard to nervous diseases in a strict sense, and never in the way it or its adjective is used when applied to the symptoms of a hysterical woman, or to minute or transitory changes of structure. (See an able paper by Dr. Allchin, "Westminster Hosp. Rep.," Vol. II.) I must now define the term. The function of organic matter and par excellence of nervous matter, to which I confine further remarks, is to store up energy (assimilation of material having potential energy—anabolism; decomposition of material — katabolism — with liberation of energy), to liberate that energy (nervous discharge) at different rates against resistances of different amounts; the energy liberated is dissipated or does work, or there are both consequences according as the resistances encountered are or are not overcome, or are only partially overcome. We have here to deal with abnormalities of this function—that is, with abnormal physiological states (I need not always add the adjective abnormal).

There are two kinds of physiological or functional states in cases of disease of the nervous system. (1) Function may be exalted, and is sometimes, as that of cells of a discharging lesion, very greatly exalted; these are superpositive functional changes—that is, hyper-physiological states. (2) Function may be diminished or lost; these are negative physiological or functional states—they are hypo-physiological states. Destructive lesions come in this category, although there is in them loss, not merely of function, but of the functional material also. We shall have to speak later of a negative functional change in temporary post-epileptiform paralysis; that negative change (there is temporary exhaustion of nervous elements) is a direct result of the excessive discharge in the prior paroxysm; the two opposite functional states of the same nervous elements occur in immediate sequence.

There are degrees of negative function from defect to loss. Of these I will say nothing. There are two greatly different degrees, if, indeed, we may not say two kinds, or superposi-

tive (hyper-physiological) function. We are concerned with both in this inquiry; it is imperative to distinguish them clearly. There is (*a*) the vast exaltation of function of cells of a discharging lesion. This degree is always produced by some pathological process—that is, by morbid nutrition, a nutrition which alters the composition of nervous matter. This, the first degree of functional change, is of a highly explosive character; it issues in paroxysmal discharges. There is (*b*) a comparatively slight exaltation of function with which we shall be particularly concerned when we consider post-epileptiform conditions. In these conditions, besides paralysis answering to a negative functional state of fibres of the second segment of the kinetic route, there are increased tendon reactions answering to a hyper-physiological state of this degree (*b*) of cells of anterior horns (lowest motor centres). For the present I shall speak only of the first degree (*a*) of superpositive functional states—that which is of a highly explosive degree, and is produced by a pathological process.

The hyper-physiological state (degree *a*) in epileptiform seizures is what I call a "discharging lesion." It is a crude hyper-physiological state just as its diametrical opposite, loss of function, is a crude hypo-physiological state. I continue to speak of healthy cells as stable; they are naturally unstable of course, but I mean by using the term stable that they are so in comparison with those of discharging lesions, which I speak of as being highly unstable. The discharging lesion is of a few cells which have got far above the rest of the cortical cells in degree of tension and instability of equilibrium. That lesion is made up of cells of nervous arrangements which represent some most special movements of a particular muscular region; the sudden and excessive development of these movements from discharge of those cells is the convulsion incipient ("signal symptom" of Seguin), or, if there be no spreading of the spasm, it is the convulsion total.

The hyper-physiology of epileptiform seizures is the element of the threefold clinical problem of which we are sure. We are quite certain that normal movements are the results of liberation of energy attending katabolism; thus the inference is irresistible that that sudden and excessive development of many movements at once, which we call convulsion, must be a result of an excess of the same physiological process—of a

sudden and excessive energy-liberation attending great and rapid katabolism. I will give illustrations of the different effects of discharges of stable cells (cells of comparatively low tension and of comparatively stable equilibrium) in normal operations, and of those of cells of a discharging lesion (cells of very high tension and very unstable equilibrium) in a convulsion. The aim is both to illustrate what was said in Chapter I. on the differences between movements proper and convulsion, and to emphasize that abnormal states and processes in disease are departures from normal states and processes by an illustration of departure by a vast excess from a normal physiological state; the term hyper-physiological keeps us well in mind that in convulsion there is such a departure.

It would be most absurd to say that there is a buttoning centre, but for convenience of illustration we may imagine such a centre. I illustrate by motor elements only, although, of course, sensory elements are concerned in all operations. In the operation of buttoning there are slight and slow discharges of normal stable cells (fibres are understood), of different nervous arrangements of our supposed centre (lower centres and muscles being understood) simultaneously and successively; in consequence there is a harmony and a melody of the different movements of the hand and arm by which the button is got into its hole. There is harmony by contemperation (contemperation is, says the "Imperial Dictionary," an obsolete word. One of its meanings given op. cit. is, "The act of reducing a quality by admixture of the contrary") of different movements, and there is melody in that the compound contemperated movements follow one another at proper time-intervals; there is what is commonly called coördination, although the time-element (melody) is not much considered in most accounts of the process of coördination. So much for the discharges of stable cells in the operation of health.

Now, let us suppose that the cells of the buttoning centre become by some, any, pathological process, so highly unstable as to constitute a discharging lesion. (For the present we shall speak as if the excessive discharges of the highly unstable cells did not provoke similar discharges of stable cells of collateral centres.) The discharges are sudden and rapid (I do not mean by using the word rapid, here or anywhere,

that nerve impulses travel more rapidly in the case of excessive discharges, but that more occur in a given time than is normal—that there is a greater quantity of motion in a given time) and of all the cells nearly at once; they do not cause a speedy and vigorous act of buttoning, but a short, severe, and rapid contention of all the movements of that operation; this contention is convulsion of the hand and arm. There is no harmony and melody; on the contrary, in the tonic stage all contemperation is lost and all time-intervals are merged, so that there is but one rigid state of the musculature of the hand and arm, and, in the clonic stage, there is but a succession of such rigid states. There are no movements properly so called in this convulsion; but, if I may use the word for once, there is first but a single big useless movement, and next a series of such so-called movements which do nothing but "mark time."

The illustration, although the supposition of a buttoning centre is grotesque, may render clear what presumably occurs when A. is "attacked by his convulsion." The convulsion is a brutal development of that man's own movements. I make this odd remark because I do not think that it is always vividly realized that an ordinary severe epileptic "attack" (to take that kind of fit for a moment's illustration) is nothing more than a sudden excessive and temporary contention of very many of the patient's familiar normal movements—those of smiling, masticating, articulating, singing, manipulating, etc. A convulsion is not something altogether *sui generis.* Speaking figuratively and more generally, and still of an epileptic paroxysm, there is the mad endeavor of the highest centres to develop the maximum of function of every part of the body, animal and organic, and of all parts at once; the phenomena of a very severe epileptic fit show that this endeavor is nearly successful; the patient is almost killed by the paroxysm, and is nearly dead (deeply comatose) after it.

I repeat that the cells which have become very highly "explosive" (those of the discharging lesion) continue to be elements of the same nervous arrangements, and that these nervous arrangements are still connected in the very same ways with the remainder of the nervous system as when their cells were stable. (There is the obvious qualification that some nervous elements may have been destroyed by the same

pathological process which also caused high instability of other elements, and thus that some connections of the latter are cut.) Hence I do not speak of the highly explosive cells as being in any motor centre, but as being of that centre. I submit that the highly "explosive" cells of a discharging lesion will on their fulminating discharge overcome the resistance of, and thus produce excessive discharge of, collateral stable nervous elements. The epileptiform fit (excepting, perhaps, some slight terminal fits) is not the result only of the discharge of the cells of the discharging lesion, as was supposed for a limited illustration, when speaking of the imaginary "buttoning centre." Making a purely arbitrary limitation, we may imagine that the primary excessive discharge, that of the fulminate of the thumb centre, only produces directly the initial spasm, that of the thumb (the "signal symptom"); all the rest of the convulsion will be indirectly produced by it, by compelled excessive discharges of stable cells. Of these compelled discharges I shall speak again later.

It will now be seen why the term physiological fulminate is occasionally used. I use it in almost a literal sense; the discharging lesion is supposed to be a detonator of collateral stable cells, just as a fulminate (in the artillerist's use of the term) is of the comparatively stable gunpowder in a cannon. The only thing persistently physiologically abnormal in the patient we will call A. is that some few cells of his thumb centre have become fulminant. To speak figuratively, this "mad part" compels many collateral "sane" cells and cells of middle and lowest motor centres, and ultimately the muscles, to coöperate in its occasional and sudden excesses—makes them "act madly" for a time. If the few highly explosive cells, those of the discharging lesion, could be destroyed, the patient would be rid of his fits; he would lose nervous elements which are doubtless never of value for coöperation with the collateral stable cells in normal operations: he would lose cells of negative value and of positive injury—cells like those of an animal poisoned by strychnia, which on their discharge, run up movements into useless contentions. The cells of A.'s fulminate, when called on to coöperate with normally stable cells in any operation, function excessively, and so as, after the manner of a detonator, to cause wide excessive discharges of many stable cells. It is a pity that A. cannot be rid of these

worse than useless cells; but I know of no way of effecting this riddance. There is the surgical question of cutting out part of the cortex.

I have in the foregoing used, regarding hyper-physiological discharges, the term "excessive," and, regarding the resulting convulsion, the term "severe;" the more excessive the discharge the severer the fit. I now use more precise terms, and consider this part of the subject as it was in effect, so it seems to me, long ago considered by Herpin. I regret greatly that my ignorance of physics renders me unable to deal with it adequately.

With regard to nervous discharges, or as I shall here say, liberations of energy by nervous elements, we have to consider two aspects—quantity of energy liberated, and the rate of its liberation; the two varying factors both in normal and in "excessive" nervous discharges. With regard to the convulsion, we have to consider its degree, and the rate with which it is produced. (Herpin, in his valuable work on "Epilepsy," sums up several propositions as follows (italics in original): "En résumé: *plus le début est long, moins la crise est violente, plus il est instantané, plus l'accès est intense.*") We have to study the amount of convulsion, the range of convulsion, and particularly the "deliberate" or "sudden" rate of onset. In two liberations of equal quantities of energy at different rates there is the same momentum or quantity of motion. But the force of the more rapid but shorter liberation of energy will be greater than that of the slower and longer liberation. Force only exists while it lasts; there is no doctrine of conservation of force. The more rapid the liberation of energy by a discharging lesion, the greater resistances will be overcome, the more numerous collateral stable elements will be compelled to discharge, and thus the more the amount of convulsion and the greater its range.

I suppose that there are degrees of instability of the cells of the fulminates in different cases of epileptiform seizures and at different periods of the same case. When A.'s fits "get worse" (greater amount of convulsion and greater range of convulsion), more cells may have then become highly unstable, or those already highly unstable may have become still more so; his fulminate becomes more fulminant, but that fulminate is made up of cells of the same part of the "motor region," if

not of the very same cells. I now consider, it must be taken hypothetically, the differences of fulmination of discharging lesions. Let me suppose two cases, in each of which there is a discharging lesion constituted by cells of the thumb centre. In the patient A. the cells are (I am only able to distinguish vaguely) of lower degree of high instability (his fulminate is much less of a fulminate) than in the case of A. A. In both the first spasm is of the thumb. I shall mention the cortical discharges only; the sequent discharges of the lowest motor centres and of the muscles are to be taken for granted.

In A. the primary discharge, that of the fulminate, is deliberate, produces (secundo-primary) discharges of few collateral stable cells, and produces them slowly one after another; the corresponding convulsion sets in deliberately and spreads slowly; is of little range (very local; for example, of the arm only), is of long duration, and there is an easily traceable distinct sequence over the range (all this, of course, comparatively with what occurs in the next case). In A. A. the primary discharge is sudden, produces (secundo-primary) discharges of many collateral stable cells, and produces them rapidly and more nearly at the same time; the corresponding convulsion sets in suddenly and spreads rapidly; it is of great range, is of short duration, and there is a less easily traceable distinct sequence over the range.

The more rapid, though the shorter, of the two primary liberations by the discharging lesions—supposing an equal quantity of energy to be liberated by each—will overcome greater resistances, and will thus compel discharges of a greater number of collateral stable elements; the convulsion produced will be both of greater amount and also of greater range. Lines of many different degrees of resistance will be overcome by such a primary liberation more nearly at the same time; the convulsion will more quickly attain its maximum at every part affected, and will be more nearly of the same degree in all parts affected. . . . Returning to epileptiform seizures, compound order is observed on a small scale in fits involving limited regions of the body (very well seen in the face), and on a large scale when such seizures become universal. There is a very intricate compound sequence from the beginning of the fit to its universalization. I shall, however, make artificial separations, and say of a fit which becomes

universal that excessive discharge beginning in some cells of a part of the right middle motor centres produces convulsion —(1) starting in the left hand and spreading up the arm and down the leg (first side), (2) involving both sides of the trunk, and (3) finally gaining the limbs of the right (second) side when all parts of the body are in convulsion together. That the left limbs are convulsed from discharge of the right middle motor centres, by intermediation of the first set of fibres of the second segment, is not doubted. I used to suggest that convulsion of both sides of the trunk and of the right limbs is also produced by discharge of that half by the second and (or rather as I should now say) third sets of fibres. This was rash. For, supposing that some convulsion of the second (right) side is producible by discharge of that one half (right), it does not follow that the convulsion which actually occurs is produced by it alone. For my hypothesis of representation of both sides of the body in each half of the brain it would suffice if there were any degree of tonic or clonic convulsion of the right (second) side. For I only suppose that the right side is represented less than and subordinately to the left side, and also second in time in the right half. The subject is a difficult one. Horsley finds that when the corpus callosum is divided, excitation of the right (I continue to say "right" for convenience) middle motor centres produces epileptic convulsion of the left limbs only. In the case of dogs, Franck and Pitres found that artificially induced discharges of the right "motor region" caused universal convulsion when the left motor region had been extirpated and the corpus callosum divided. I quoted them to this effect in my third Croonian Lecture.[5] They, however, as I then said, attributed the universalization of the convulsion to the pons, medulla, and cord (which together I call the lowest level).

Considering, then, the opinions of those who hold that the second and third sets of fibres, as well as the first set (all the fibres of the second segment of the kinetic route interconnecting the right middle motor centres with the lowest motor centres), end in the left lowest motor centres, the researches of Franck and Pitres and those of Horsley, and also the great complexity of the subject, I ought not to be dogmatic as to the process by which universalization of an epileptiform convulsion is produced. Most likely in severe seizures the left

middle motor centres are discharged, as Horsley supposes, after the right by intermediation of callosal fibres, although possibly some slight convulsion of the right limbs is produced from discharge of the right half alone by intermediation of the second and third sets of fibres. And most likely, too, the anterior commissures of the pons, medulla, and cord (intrinsic fibres of the lowest level interconnecting left and right lowest motor centres) are concerned. Probably the loss of consciousness answers to excessive discharges of the highest centres caused by the medium of sensory (upward) fibres. The process of universalization of epileptiform seizures is a very intricate one, and deserves more precise analysis than I am capable of making.

I stay here to say that, taking the case of A. in illustration, when we have located his lesion we have done anatomical work only; when we have noted all we can about his convulsion (therefrom inferring that the lesion is a "discharging lesion" and the effects of its discharge direct and consecutive) we have done physiological work only. The experimenters on animals do the anatomico-physiological work thoroughly well.

Taking up again the case of A., we have now the third element, pathology, to consider. Here is the great difficulty. Being sure that A. has a discharging lesion of his thumb centre, there may be no evidence to show whether that lesion is produced (indirectly) by tumor or not. Here I urge again the necessity of distinguishing between the physiology and pathology of cases of nervous disease. Having urged that there is a degree or kind of functional change (*b*) which is not the result of any pathological process, I am all the more wishful to urge the distinction. I continue to speak of the first degree (*a*), and urge the distinction from this point.

It may be epigrammatically said, and with truth, that an epileptiform seizure is "only a symptom." Nevertheless it is always symptomatic of one physiological thing, a "discharging lesion." Otherwise put, an epileptiform seizure is not a symptom of tumor, of "softening," or of meningitis: it is a symptom of the one thing, high instability of certain cortical cells, however produced—produced by any pathological process. Physiologically speaking, there is but one "cause" of epileptiform seizures—viz., high instability of some cells of some centre of the Rolandic region; but there are many

"causes" of them if we mean pathological processes leading to that instability. So that in the case of A. (he being subject to fits), the question in pathology is not the vague one: "What is the disease of a certain part of his cortex?" but "What abnormality of the nutritive process has produced such an alteration in the composition of the material of cells in that part as to render them highly explosive, and how was that abnormality set up?" Or, more generally, and regarding all cases of epileptiform seizures (and epileptic, too), we put the question: "How are local persistent discharging lesions established and kept up?"

The first question is: "What is the most general nature of the abnormal nutritive process of cells of discharging lesions in epileptifom seizures?" This we ask ourselves, whatever the particular gross pathology or morbid anatomy, tumor, "softening," etc., may be in any case.

I have so often spoken on this subject that I will only mention the hypothesis that the nutrient fluid bathing the cells is comparatively stagnant, and that in consequence there is inferior nutrition. I suggest that there is "substitution nutrition," phosphorous compounds becoming more nitrogenous, or nitrogenous compounds more nitrogenous. I may illustrate by the well-known case of glycerin becoming explosive (nitroglycerin) when some of its hydrogen is replaced by nitric peroxide. The composition of this substance is altered, but its constitution remains the same. It may possibly be that the mass of the explosion of nerve cells, both in health and convulsion, is of non-nitrogenous matter, as is the case in the normal discharges of muscle. But the suggestion is that a nitrogenous substance is the "pivot" of the metabolism of nervous matter both in physiological and in hyper-physiological states. ("Whether the chief product of the metabolism of any tissue be a proteid substance, or a fat, or a carbohydrate, proteid substance is the pivot, so to speak, of the metabollism, and nitrogenous bodies always appear as the products of metaboism."—Foster, "Physiology," Part II., p. 828.) I suppose the greater nitrogenization of the material of nerve cells makes it highly explosive, but that the constitution of that material and the morphological structure of the cells remains the same. If this hypothesis be valid, there are presumably degrees of substitution (as there are in the three

chloracetic acids, for example), and consequently degrees of high explosiveness. It may be that when a patient's fits "get worse," the original fulminate becomes more fulminant by still greater nitrogenization, as well as that more cells become part of his fulminate. The abnormal nutrition leading to change of composition is a pathological process, while the high explosiveness it produces is a functional change. Surely it is the change of function which should be called a functional change. Whether these speculations be valid or not, it is a warrantable inference that the nutrition of the cells of the discharging lesion goes on, that it goes on wrongly, and that some material of the cells does become highly explosive.

Now I consider differences in sizes of cells with regard to their normal and morbid nutrition.

For theoretical reasons, and after the observations of Betz and Mierzejewsky, I suggested[6] that those centres of the motor region which especially represent small muscles (eyes, face, and hands) will have a greater number of small cells than those which especially represent the large muscles of the limbs. I think this is generally true of the "motor region." And I suggest that it accounts for the great frequency of onset of epileptiform fits in the hand and face, for reasons to be given presently. The "leg" centre contains many large cells, but it also contains some small cells. Bevan Lewis' researches, to which I am very greatly indebted, show that the parts of the motor region representing small muscles have most small cells. He says, and this bears on the remark I made on the leg centre, that "alongside the largest pyramidal cells are numbers of others of the *smallest* dimensions" (italics in original),[7] so that the discharging lesion of the "leg centre" may be made up of small cells.

Although it is convenient for some purposes to say "small muscles," the expression is not exact. For I urge once more that nervous centres do not represent muscles, but movements. I will substitute the expression "small movements" for "small muscles," and that of "large movements" for "large muscles." As these terms, the best I can think of, are vague, their meaning must be taken as defined here. Most of the movements of the hand are "small" according to the definition I now give of "small movements." The parts moved have little mass, and in most of the operations they serve in,

there is little more added mass. The muscles are small and numerous, and in most operations by the hand their movements are of little excursion, of short duration, and rapidly changing. Short, prompt, and frequent liberations of small quantities of energy will be required for these successions of "small" different movements; I submit that the nervous arrangements for these movements of the hand have small cells, and very many small cells. Most of the muscles of the shoulder are "large movements," according to the following definition. The muscles are large and few; there is much mass to move, the whole arm to lift, and the added mass is often great in some of the operations they serve in, as in lifting weights the hand takes up. In most operations they serve in, the movements are of large excursion, of comparatively long duration, and are comparatively little changing. They will require persisting and slow liberations of large quantities of energy by, I suggest, comparatively few and large cells.

A principle of representation is here in question; we have not only to do with sizes of cells, but also with numbers of cells, and therefore with the volume of different centres. Schroeder van der Kolk, illustrating by the case of the sturgeon, which has large muscles and few cells in its spinal cord, pointed out that there is not a mere relation between quantity of gray matter and size of muscles, but that the gray matter is greater in proportion to the complexity of movements of muscles. This shows that we have never to forget that centres represent movements of muscles, not mere muscular masses. I have several times drawn attention to Herbert Spencer's statements bearing on this question.[8] One remark he makes is: "In proportion to the number, extensiveness, and complexity of the relations, simultaneous and successive, that are formed among different parts of the organism, will be the quantity of molecular action which the nerve centres are capable of disengaging." Spencer takes count of both impressions and movements; I am illustrating by movements alone. The much greater volume of the middle motor centres is in accord with the fact that they represent vastly more numerous different movements than the lowest motor centres do; the muscles represented by both levels are the same, being in each case all the muscles of the body; hence the middle motor centres

contain many more cells and fibres than the lower centres do. The supposition is that parts having many small and greatly changing movements are especially represented by small cells and by many small cells, and that parts having but few and little changing ("tonic") movements are represented by large cells and by few large cells.

The size of cells is a very important matter with regard to their nutrition. Both in health and in disease small nerve cells will be nourished more quickly than large ones when both are bathed in the same nutrient fluid. ("Other things equal, the smallest cells will soonest become unstable. A mass of nervous matter in many small cells will 'present a much larger surface to the contact' of nutrient fluid than the same mass in a few large cells."—Harveian Lectures, "Med. Times and Gaz.," January 11th, 1879. My attention was first directed to this subject on reading Spencer's "Biology," where he expounds his theory of growth. A brief statement of Spencer's theory will be found on p. 220 of "The Evolution of Sex," by Geddes and Thomson, from which I quote: "In spherical and all other regular units the mass increases as the cube of the diameter, the surface only as the square." I refer the reader to Ross' great work, "Diseases of the Nervous System," Vol. I., p. 13, where he deals with the significance of differences in sizes of nerve cells with regard to normal and abnormal nutrition and its consequences. From diminished nutritive supply the small cells will atrophy sooner than large ones.) The smaller cells will become highly unstable sooner than large ones during morbid nutrition. It certainly is the fact that most epileptiform seizures begin in parts having "small movements," in parts represented by areas of the cortex having most small cells.

Four hot iron balls will become cold much sooner than the same mass of iron in one ball having the same quantity of heat as the four balls together have. From discharge of four small cells which are together equal in mass to that of one large cell there will be, I submit, a liberation of energy in a shorter time than by the large cell, supposing equal quantities liberated in the two cases. Hence another reason for fulmination of the highly unstable cells of discharging lesions if these cells are small.

To repeat several hypotheses, epileptiform seizures begin

most often in parts of the body having "small movements;" these movements are represented by nervous arrangements having many small cells. Small cells present a more extensive surface to nutrient fluid than the same quantity of gray matter in large cells, and will be more quickly nourished than large ones are. Nerve cells become highly unstable from an abnormal nutrition; such that, although their structure and the constitution of their material are unaltered, that material becomes of more nitrogenous composition, and thus more explosive. Small cells become highly unstable more readily than large ones do; thus discharging lesions are supposed to be especially of small cells. A rapid liberation of energy overcomes greater and more numerous resistances than a slower liberation of an equal quantity of energy. Small cells liberate their energy in a shorter time than large ones; hence the currents developed by fulminates of small cells overcome greater and thus more numerous resistances than would fulminates of large cells, and hence produce more convulsion and greater range of convulsion.

We have assumed that the nutrition of the cells of the discharging lesion is continuous, and have supposed in effect that the nutrient fluid is comparatively stagnant. But how is this comparative stagnation brought about? This brings us to pathology, commonly so called. We have to distinguish between what I may call the coarse pathology of a case and its immediate pathology.

CHAPTER III.

THERE is not rarely after epileptiform seizures local temporary paralysis, and sometimes aphasia with it. For the present I speak of paralysis only. In this inquiry we have no direct concern with paralysis, temporary or permanent, owing to destruction (by clot, softening, or any other pathological process) of any part of the brain, not even if the malady set in with a convulsion.

The term "post-epileptiform paralysis" will be used to include all paralyses the immediate sequels of epileptiform seizures. I hold the hypothesis (essentially that of Todd and Alexander Robertson) that there is exhaustion of central nervous elements, including fibres of the second segment of the kinetic route, and that this is produced by the sudden and excessive discharge in the prior paroxysm. These nervous elements are exhausted, not otherwise injured, so that recuperation is prompt, and the corresponding paralysis is temporary. There is a sequence of two opposite functional states, superpositive of the first degree (*a*) in the paroxysm, and then after the paroxysm negative. But there is often more than a negative functional state in the post-epileptiform condition. We may find increased tendon reactions implying a superfunctional state of the second degree (*b*) of lowest motor centres. The duplex nature of these post-epileptic conditions must be borne well in mind.

I will now give an illustration of post-epileptiform paralysis. It may be taken to be an artificial one, but it is essentially that of a patient whose case I reported.[9]

A man, B., was seemingly (1) quite well when he arrived at my house after a walk of about a mile. (It is convenient to speak of this as the "first stage," not of course of the fit, but of the dramatic occurrence I am relating.) (2) (*a*) A fit began in the toes of the left foot. (Referring to the real case

[op. cit.], the patient said that in all his seizures the first "sensation" was to the outer side of the great toe; very likely I missed a very earliest stage, one possibly of spasm limited to the ball of the big toe.) (*b*) The whole leg was gradually involved, the spasm passing up the limb. In about eight or ten minutes the convulsion ceased, when (3) the leg was found to be (*a*) paralyzed, and (*b*) there was exaggeration of its knee-jerk and clonus of the foot. (4) In six hours or less he was seemingly well again, certainly he was then rid of his paralysis; and next day (I did not test his tendon reactions before) his knee-jerks were normal, and there was no foot clonus. Let me consider the four acts of this drama.

(1) The patient was not really well when he arrived at my house. He had a persistent discharging lesion, presumably of a few cells of his leg centre (perhaps only of some of those of the hallux centre); so to say, he always carried it about with him, or, to speak more precisely, it was a persistent quasi-parasitical hyper-functionable part of himself.

(2) (*a*) The discharging lesion did function and that excessively, and possibly produced the initial spasm (of the toes) by its sole discharge: but (*b*) next, as a fulminate, by overcoming the resistance of (discharging) other cells of the leg centre it compelled these normal stable cells to discharge—compelled them to coöperate in its excess. So much for the discharges (primary and secundo-primary) of the cortex (middle motor centres). That there was a supernormal activity from cortex excessively discharging to and of the muscles of the leg convulsed is certain. The route of the numerous and rapidly succeeding nerve impulses from the hyper-physiological cortical process would be certain fibres of the corona radiata, of the internal capsule, of the crus cerebri, pons, and medulla of the right side, then of the fibres of the left lateral column (possibly also of the second and third sets of interconnecting fibres, both of which I shall ignore here) up to the anterior horns (some lowest motor centres); so far the second segment of the kinetic route is concerned. The resistance of the cells of these lowest motor centres would be overcome (secondary discharges), and the impulses (presumably increased in number) would then be of fibres of the nerve roots from those centres, thence of the continuing fibres of nerve trunks and their branches to the end plates of the muscles (third segment of the

kinetic route); the resistance of the muscles would be overcome, and there would finally be great "explosive decomposition" of muscle substance (tertiary discharges).

(3) The paralysis signified exhaustion of nervous elements previously excessively functioning in the paroxysm. There would be exhaustion of cells of the cortex, not only of those of the discharging lesion, but also of those collateral stable cells which it, as a fulminate, compelled to discharge excessively. There would be exhaustion, too, of the fibres passing down from both sets of cells. The hyper-kinetic route, or at least part of it, would now be a hypo-kinetic tract. There would be exhaustion as low down as certain lowest motor centres; these centres were therefore exalted in function (second degree of superpositive functional change), hence the increased tendon reactions.

(4) The paralysis had passed off; the patient could walk well. Next day the knee-jerks were normal. There was recuperation of the exhausted nervous elements. No doubt the cells of the discharging lesion (after the fit, stable below normal) began at once by gradual morbid nutrition to re-attain high tension and very unstable equilibrium—"began to prepare for the next fit."

So far as I have seen, post-epileptiform paralysis is always most of the parts which were first and most convulsed in the preceding paroxysm, as in the case of the patient B. This correspondence is denied by some eminent physicians; further observations will settle the matter.

I think there is a relation of proportionality between the severity of the epileptiform fit (severity of the discharge) and the subsequent temporary paralysis (amount of exhaustion). I should have thought this a truism if it had not been expressly denied. It is, however, averred that while after the epileptiform kind of seizure there is often decided paralysis, there is not, as a matter of fact, any whatever after the severer fits of the epileptic kind. I demur to the "fact," believing that while after a limited epileptiform seizure there is often great local paralysis, loss of many movements of a small part of one side of the body, there is after a severe epileptic fit widespread paralysis, loss of few movements of most if not of all parts of both sides of the body. Indeed, I submit that the whole con-

dition of bodily impotence after a severe epileptic fit is paralysis, and that, speaking generally, there is really more paralysis than is found after severe epileptiform seizures.

The relation of an epileptiform fit to the sequent paralysis, or, more exactly, the relation of the two opposite functional states, discharge and subsequent exhaustion, is not a simple relation. According to the hypothesis I hold there should always be some, however litle, paralysis after an epileptiform seizure, at least some in the parts first and most convulsed, however slight the attack may have been. Trifling degrees of paralysis are easily overlooked. This deserves remark.

After a slight epileptiform seizure starting in the hand, the patient may have difficulty in picking up a pin and may yet be able to grasp strongly. There is clumsiness of movement, or perhaps some may say "loss of muscular sense." These terms must not let us overlook the fact that the slightly abnormal motor condition of the hand is a double condition, one of two opposite elements. There is in the case supposed difficulty in picking up the pin, because a few most special movements of the hand are lost (so far paralysis, negative element); the pin has to be picked up by the next most special movements remaining, which are now the most special of the movements (positive element); they do not serve so well as the most special would have done; hence the operation is "clumsy."

From such quasi-trifling paralysis as that indicated, loss of a few most special movements of the muscles of the hand, there are met with after severer epileptiform seizures beginning in the hand degrees of loss of next and next most special (or, equivalently, of the next and next more general) to loss of all movements of the muscles of it, and of some movements of the rest of the arm too; there is then no "clumsiness," for no movements of the hand remain to do anything.

It will be well to draw attention to resemblances and differences between post-epileptiform hemiplegia and the hemiplegia which is the result of destruction of the motor part of the internal capsule. For the moment assuming that the explanation given of the former is correct, and that the statements accepted by most physicians as to the latter are accurate, I will call the former Hemiplegia E, the latter Hemiplegia D.

First, for resemblances, taking to begin with, for compari-

son and contrast, recent Hemiplegia D. The regional (paralytic) affection is the same as in Hemiplegia E, and in each depends on a negative state of the same strand of fibres, those interconnecting the right middle motor centres and the left lowest motor centres. (I shall speak only of what I called the first set of fibres.) Now for differences. In Hemiplegia E (as in the case of the patient B.) there is exaggeration of the tendon reactions; in Hemiplegia D they are, we shall suppose, normal. Correspondingly for the negative lesions, in Hemiplegia E the negative lesion is of the whole length of the interconnecting fibres mentioned (and spoken of before as the second segment of the hyper-kinetic route become a hypo-kinetic tract) up to, but not involving, the lowest motor centres. In recent Hemiplegia D the negative lesion is quite local, a breaking up of the capsular part of the interconnecting fibres; the rest of the segment, its whole extent below the capsular break, being normal.

Now for old cases of Hemiplegia D. In these there are increased tendon reactions. In them a negative lesion secondary to the capsular one has been established; there is then a negative state (destruction) of the fibres (lateral sclerosis) below, and added on to, the local capsular lesion. (Sherrington has traced the "descending wasting" in the lateral column of the cord of the monkey as low as the coccygeal nerve roots.) That is to say, in the case of Hemiplegia E there is established at once the state of things which only occurs late in Hemiplegia D. So long as the exhaustion lasts (so long as the central kinetic route is hypo-kinetic) in Hemiplegia E, the situation is the same as in old cases of Hemiplegia D; it is the same for the time being whether the nervous elements in question are functionally dead, as in the former, or actually dead (broken up and wasted away), as in the latter.

In a popular use of the word "cause," these negative conditions may be said to cause the increased tendon reactions, just as, popularly speaking, opening flood-gates is said to cause water to flow. But using the word cause properly, it is an impossibility that the negative functional state in either Hemiplegia E or D can cause anything; a nothing cannot cause the something, the superpositive condition for exaggeration of the knee-jerks.

It is agreed upon that the increased tendon reactions in

cases of hemiplegia depend on exalted "excitability" (a superpositive functional state) of nervous elements of some anterior horns. There are different hypotheses as to the process by which that exaltation is produced, and thus as to the exact state of the cells of the anterior horns concerned. Two questions may be asked: Is the abnormal condition of the cells the result of any pathological process involving the horns themselves? Or, are the cells healthy and only in greater activity? I reply to the first question negatively, and to the second affirmatively.

I take for further consideration an old case of Hemiplegia D, one in which lateral sclerosis is established. And now I consider rigidity as well as increase of the knee-jerk (limiting illustration to that one tendon reaction), both of which superpositive phenomena it is agreed upon imply exaltation of function of cells of anterior horns. One hypothesis as to the production of exaltation of function of the anterior horns (some of the lowest motor centres) is that the same process which leads to destruction of fibres of the lateral column produces, when come to the horns with which they are in physiological union, increased "excitability" of their cells; the same process, on this hypothesis, produces loss of function (destruction) of fibres and its physiological opposite superfunction of cells.

This hypothesis cannot apply to Hemiplegia E; in this case it is not likely that there are caused by the excessive discharge a negative state of fibres, and a superpositive one (excess of the normal) of cells. All the more that after some epileptic fits there is found exaltation of the knee-jerk, and after others loss of it; differences, explainable on differences, in the severity of the discharge in the paroxysm, and on the different "depths" of exhaustion after it; there is a negative functional state in both, but lower down in the kinetic route, of cells of anterior horns, when the knee-jerks are absent.

My hypothesis is that both in Hemiplegia E and in old cases of Hemiplegia D the cells of the anterior horns are healthy. They are, I submit, untouched by the pathological process causing the lateral sclerosis or by any other, and they are simply over-active, partly from loss of control, and partly from non-antagonized cerebellar influx (second degree [*b*] of superpositive functional change). But I do not state the hypothesis of loss of control as I used to do. There is sometimes

exaggeration of the knee-jerk in recent cases of hemiplegia, and this may be owing to sudden loss of cerebral (middle motor centres) control. I shall, however, speak as if the current opinions were correct—that both the increased tendon reactions and rigidity "wait for" the descending degeneration in cases of Hemiplegia D. I now adopt an hypothesis put forward by Gowers, which is to the effect that close upon the anterior horns there are small inhibitory centres, the anterior horns being, in his nomenclature, "muscle centres."

In cases of recent Hemiplegia D, before the degeneration in the lateral column, these local inhibitory centres are intact, and the knee-jerks are not exaggerated. But I suggest that in such cases of Hemiplegia E, as that of the patient B., they are exhausted, as well as are fibres of the lateral column; after those epileptic fits, when the knee-jerks are lost, both the inhibitory centres and the muscle centres are exhausted. Now, taking old cases of Hemiplegia D, and dealing with rigidity as well as with the exaggerated knee-jerk, I would say that the "descending" process, when it reaches the anterior horns, destroys the small local inhibitory centres; the anterior horn proper, the "muscle centre," now bereft of its inhibitory centre, is uncontrolled. The cells of the "muscle centres" are not invaded by any pathological process; they are healthy, but yet over-active from loss of control. This hypothesis as to old cases of Hemiplegia D invokes but one mode of action —destruction. That hypothesis brings the facts of Hemiplegia E into harmony with those of long-standing cases of Hemiplegia D; it accounts for the absence of increased tendon reactions in recent cases of Hemiplegia D. Certainly cases of Hemiplegia E have to be considered as well as cases of recent and old Hemiplegia D in all hypotheses as to the nature of increase of the knee-jerk in cases of brain disease.

In some cases of epileptiform seizures at, or close upon, their onset, there is loss, or at least defect, of speech with very little local spasm. (We have, of course, no concern in this inquiry with any cases of *le petit mal*.) I shall, however, speak only of temporary abnormal affections of speech, not in, but after the paroxysms—of what is present when all convulsion is over. ("There is a peculiar class of cases of epileptic hemiplegia in which the exciting cause of the epileptic fit at the same time damages or greatly injures voluntary power

and speech."—Todd, "Nervous Diseases," Lecture XV.) I explain post-epileptiform defects of speech in the same way as I did post-epileptiform paralyses, supposing there to be in the former exhaustion of nervous arrangements of "Broca's region." Temporary defect of speech after epileptiform seizures beginning in the (1) right hand, or (2) right side of face or tongue, or both, is not rare.

I have not observed post-epileptiform loss of speech, but only defect of speech ("partial aphasia"). I shall use the term Defect of Speech E, or Partial Aphasia E. The term "defect of speech" is equivocal, as is also the term "partial aphasia" it really covers two opposite elements, negatively loss of some speech, and positively retention of the rest of speech. (My present concern with aphasia is only a very limited one. I need not always supply the obvious qualifications to statements made in the text. The expressions "loss of some speech" and "retention of the rest of speech" must not be taken literally, as if they meant that the patient had lost certain words or propositions of the [his] English language altogether and had retained the rest of them intact. Such is an impossible condition of things when an aphasic patient corrects his mistakes. But the statements may be taken literally for the mere purpose of a limited illustration. I do not suppose that there are fixed nervous arrangements—some for these words or syllables [properly movements corresponding to syllables] only and some for those only. I would, rather than hold this mechanical doctrine, go to the other extreme, and say that there are no nervous arrangements or movements in any centres except at the time when these and those motor nervous elements are functioning together in a particular temporary grouping.) The patient gets words out clearly and may even get out simple appropriate replies, such as "Very well," and may answer correctly by "Yes" or "No," or both. This is the positive element: it is the inferior speech to which the patient is reduced. But he is not able to reply correctly except by "Yes" and "No," and by other simple and very general expressions; he is not able to converse properly on simple subjects; he is unable to explain anything at all complex. These statements give the negative element: the speech lost. It is of extreme importance to distinguish the two opposite elements of the symptomatic condition. It is

plainly impossible that the post-epileptiform exhaustion (*vide supra*), which I suppose there is of part of Broca's region, can answer to the patient's utterances—to the positive element: that negative functional state of nervous elements of the region answers to the patient's loss of some speech. His utterances, positive element, however inferior as speech, answer to activities of nervous arrangement of Broca's region which are healthy—which are in all ways normal except possibly for slight "loss of control."

We must bear well in mind that speech is a psychical process. Nowhere is it more important to distinguish the psychical from the physical. In the case of Defect of Speech E our ultimate concern is with the two opposite functional states of motor elements of Broca's region, which answer respectively to the two opposite psychical elements of the double symptomatic condition, which is unfortunately named (defect of speech or partial aphasia) after the negative element only. The motor elements alone of the sensori-motor substrata of speech are damaged. I submit that in Defect of Speech E there is correlative with its negative psychical element exhaustion of some nervous arrangements of Broca's region, which is productive of loss of some of the complex movements of the muscles of the tongue, etc. ("articulatory muscles"). And I submit that the speech remaining possible is correlative with integrity of other nervous arrangements of that same region for other complex movements of the same muscles; these healthy nervous arrangements function during the inferior speech remaining possible to the patient, and produce the corresponding articulatory movements. I have now to support the conclusion that the inferred exhaustion of some elements of Broca's region in Defect of Speech E produces paralysis in the sense of loss of complex movements of the tongue, etc.

There is often with Defect of Speech E some temporary decided paralysis of (loss of many movements of) the right arm. This statement, however, although allowable clinically, is not a scientific one. We should not, in a realistic inquiry, group together a psychical symptom and a physical one as if, when so considered, they were symptoms of the same order. In harmony with what I have said, the things really comparable are respectively loss of some complex movements of the mus-

cles of the tongue, etc. (corresponding to the negative element in the defect of speech) and loss of some complex movements of the muscles of the arm (in both of movements represented by the middle motor centres). In this sense the two coexisting things are paralyses. On this basis alone is their concurrence intelligible and their comparison and contrast possible. With regard to the defect of speech, it is to be vividly kept in mind that the fellow-part of the right middle motor centres (centres of the left half of the brain) for other complex movements of the muscles of the tongue, etc., and that the right and left lower (bulbar) motor centres for simplest movements of those muscles are intact.

It will be well in further exposition to stop speaking of Defect of Speech E, and to take in illustration a simpler (although much more serious) case, one of loss of speech ("complete aphasia") from a destructive lesion—say, from softening of Broca's region. I shall suppose, however, that there is retention of "Yes" and "No." (To speak is not merely to utter words, but to propositionize. The patient spoken of in the text, although not absolutely speechless, is so, except that he has only the use of the two most general of all propositions; I say "the use of," supposing that in the case taken for illustration the patient can assent and dissent by them. If he only uttered them at random, or if they were only signs of emotion, they would not serve propositionally; they would then be of no speech value.) Here the negative element is loss of nearly all speech; the positive element being retention of "Yes" and "No" only. I shall call this case Loss of Speech D, or Complete Aphasia D; but the word loss is to be taken as qualified by "except for retention of 'Yes' and 'No.'" We shall suppose there to be, as there nearly always is, right hemiplegia.

To say that the patient Aphasia D is speechless because he has lost the memory of words is to give no explanation of his inability to speak, any more than it is an explanation of the paralysis of his right arm and leg to say that it is owing to loss of volition. A part of the patient's brain has disappeared, probably the hinder part of the lowermost (left) frontal convolution (and no doubt more brain adjacent) has been changed to diffluent stuff which is brain no longer; we shall speak of this as "destruction of Broca's region." I repeat that the lesion causes paralysis. I admit that there is no

disability in the muscles of the tongue, palate, lips, etc., in their commonplace services, as in eating, drinking, swallowing, etc.; I admit it readily, because I only mean that there is loss of the complex articulatory movements of those muscles. There is nothing discoverably wrong with these muscles except for the slight facial and lingual paralysis which is a part of hemiplegia, right or left. The case illustrates that we may have loss of some movements of certain muscles without discoverable disability of those muscles. The seeming paradox in the particular case under remark is easily explained by Broadbent's well-known hypothesis.

In our speechless patient not only are the lowest motor centres of both halves of the bulbar part of the lowest level for simplest movements of the muscles of the tongue, etc., intact, but also, what is most important, complex movements of these muscles of both sides remain represented in the right half of the brain middle motor centres. Hence, on account of those remaining representations, there is no obvious disability of the muscles of the tongue, etc., in their commonplace services. Taking the simple representation by the lowest level for granted, the assumption is that the muscles of both sides of the tongue, palate, lips, etc., are represented by complex movements in each half of the brain, though no doubt more specially in Broca's region than in the fellow-region of the right. Here is an exemplification of Broadbent's hypothesis.

So far am I from comparing loss of speech, considered as a negative psychical symptom, with the physical symptoms of bulbar paralysis, that I urge that there is not even a basis for any reasonable contrast of the two—psychical symptoms and physical symptoms are "not on the same platform." Regarding the physical conditions correlative with the aphasia and the case of bulbar paralysis, the cases are alike in that there is in each loss of movements of the same muscles; on this basis I urge their resemblance as cases of paralysis. At the very same time I urge the differences between loss of complex movements of the tongue, etc., represented by certain middle centres of but one half (left) of the brain in cases of Loss of Speech D, and loss of simplest movements of those parts represented by certain lowest (bulbar) centres of both halves of the lowest level in cases of bulbar paralysis.

Moreover, the part destroyed in the case of aphasia is

motor; no one denies since the researches of Hitzig and Ferrier that the homologous part in lower animals is motor. Hence there is nothing for destruction of it to produce in these animals except paralysis. Mark that although destruction of that part of the "motor region" on the left in man produces no disability of the muscles of the tongue, etc., of either side (beyond the slight right facial and lingual paralysis spoken of as part of hemiplegia), discharge of that part develops movements of these muscles of both sides; by subagency, of course, of certain lowest motor centres of both halves of the lowest level—develops a contention of complex and of simplest movements of those muscles. From discharge of the part on the left are revealed the movements the loss of which, upon destruction of that part, is masked by compensation by the fellow-part of the right half of the brain. Yet there may be discharge (impulses passing by route of the callosal fibres) of the fellow-part on the right consequent on the primary discharge of the left. I mention again that Sherrington has traced degenerated fibres from a unilateral lesion of the lower end of the fissure of Rolando into both halves of the pons and medulla.

Hence I conclude that the patient hemiplegic and speechless (aphasic) has not only lost very many complex movements of the muscles of the right arm and leg, but that he has also lost many complex movements of muscles of both sides of the tongue, lips, etc.; the conclusion is, that his whole physical condition is, and is nothing else than, paralysis in the sense of loss of movements.

The condition is fundamentally the same in the Defect of Speech E, but the negative element (loss of some speech) is less, and the positive element (retention of much speech) is greater. Here, again, it will be well to illustrate by the case of a destructive lesion (a much smaller one than that in Loss of Speech D), by softening, destroying but little of Broca's region: I will call this case Defect of Speech D. There is not rarely from such a pathological process a condition essentially like that in some cases of Defect of Speech E. Let us suppose that in the case of Defect of Speech D, the patient frequently makes mistakes when he talks, either by using wrong words or by being reduced to very "automatic" utterances, such as "Very well," or that he often uses roundabout expressions,

such as "It won't come out here" (which, eked out by appropriate pantomime, meant "I can't make water"). And yet, to simplify exposition, I will take the patient's saying "hat" for "carpet" only, that to be a sample of the whole of his condition. Here we see the necessity of distinguishing the two elements, negative and positive. It is certain that the softened brain (really what is brain no longer; there is a hole filled with detritus of nervous elements) cannot be the cause of the patient's saying "hat;" that is, the speech left, and is owing to activity of the part of Broca's region which remains perfectly healthy. This, his positive condition in speech, is sampled by his saying "hat." His negative condition is alone owing to "disease," and is sampled by his inability to say "carpet."

The negative element in Defect of Speech D is a minor degree of that in Loss of Speech D; the positive element is a major degree of the positive element ("Yes" and "No" only) in Loss of Speech D; in the former the dissolution of Broca's region is shallow and the level of evolution high, while in the latter the dissolution is very deep and the level of evolution very low, if there be any in that region. The two cases differ in double degree. In the Defect of Speech D there is much speech left, and there are very many mistakes; in the Loss of Speech D there is very little speech left, and there are no mistakes, the patient correctly assenting to and dissenting from (by "Yes" and "No") all statements made to him.

Now for the physical condition in Defect of Speech D. Answering to the slight negative element, there is loss of but a few complex articulatory movements represented in Broca's region; answering to the positive element, there is retention of many other such movements; or, we may say, limiting illustration to the samples, there is loss of the complex movements answering to inability to say "carpet," and retention of the complex movement answering to saying "hat." If any one says that a word (properly a syllable) is not a movement he is agreeing with me; for I am contending that activity of nervous arrangements representing certain complex movements (with, of course, corresponding impressions) of the tongue, etc., is only the anatomico-physiological process going on during the appearance of what are the psychical things—words. I need only say that the softening in Defect of

Speech D is the negative lesion equivalent to the exhaustion in Defect of Speech E. The utterances both in Defect of Speech E and D are the outcome of activities of perfectly healthy nervous arrangements. The negative lesion in both is dissolution; in each the speech, however imperfect, as certainly signifies evolution going on on a lower level, as the speech of healthy persons does evolution going on on the normal level.

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SURGICAL TREATMENT

OF

DISEASES OF THE BRAIN.

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CHAPTER I.

INTRODUCTION.

Two modern achievements have advanced and ripened the idea of curing pathological conditions in the interior of the cranium and of the brain itself through operative interference. One is the new teaching of the localization of brain functions, as distinguished from the old idea of the unity of the organ of the mind; the other is the certainty of preventing and curing purulent inflammation of the pia mater with which the antiseptic method has empowered us.

According to modern physiology, the anatomy of the brain gives rise to two fundamental laws: the principle of the localized function and that of the substituting function. Each individual function has a definite seat in the central organ from which it emerges, and other localities may take the place of those whose functions are slow or abolished. Just as the first of these principles enables us to localize the seat of a disturbance in the brain, the other allows us to wound, incise, and even remove parts of the brain without permanent abolition of the affected function. In this respect the physiological differences of the cortex of the brain are most important, since this part is encountered directly after removal of the bony walls of the cranium and is the first point of attack in every case.

Through experiments, repeated thousands of times on animals, we have learned to distinguish those localities of the cortex, which when irritated, uniformly cause contractions of the same muscles on the opposite side of the body, and of

those whose excitation is not followed by muscular contraction. The results of the study of excitations were confirmed, completed, and extended by constantly improved methods of extirpation—the so-called exclusion experiments. Certain large sections of brain-cortex may disappear without any visible disturbance; while in other portions, the smallest loss will weaken, interfere with, or abolish some motion or sensation.

In this way the surgeon has received the necessary instruction for the guidance of his knife in penetrating into the depth and in searching for definite and circumscribed areas of disease.

But the knowledge of localities and routes by which we must proceed would not have justified us in opening the closed cranial cavity if the barrier against incision down to the cranial bones and trephining the latter had not been removed. This barrier was the occurrence of purulent meningitis, which placed the patient in the greatest danger of a fatal termination in every case in which the cranial cavity had been opened through an unfortunate accident or a well-planned operation, the practitioner being unable to prevent or to cure it. It was necessary to arrive at the present state of perfection in antisepsis before surgical invasion of the skull would be rid of this drawback.

A great deal of physiological and surgical preparatory work was necessary before there could be any consideration of brain surgery and before the mechanical assistance of the surgeon could be utilized in the treatment of diseases which were until a few years ago regarded as belonging exclusively to the domain of internal medicine.

Since then, it has become the duty of surgeons to familiarize themselves more closely and deeply with the diseases of the central nervous system, so that they can examine them and regard them in the light of their own opinions.

If, in addition to the diagnosis, the advisability of operation be left to the physician, the surgeon sinks to the position of workman—a position which he held in former times when his sphere was limited to carrying out the directions given him, without any questions. In this connection, as in all others, it is desirable to examine personally before we coincide or differ with the opinions of others. The surgeon must appreciate

the conditions and relations of the procedure which he contemplates. In like manner, the physician must thoroughly understand the result and extent of the mechanical aid which he requests; in this respect there is still much to be desired. The physician only sees an absence of danger in the incision rendered innocent by the antisepsis of the surgeon, and is therefore more apt to resort to it than he who appreciates the price at which supposed safety is realized. This explains why cases are referred to the surgeon with the expectation that his operation will produce results entirely incompatible with skill founded upon scientific experience.

It has happened more than once, that well-known colleagues have requested me to operate, especially in cases within the province of brain surgery, and I had to decline. Such differences induced me to attempt to establish the relationship between physician and surgeon by presenting the position which, acccording to my opinion, surgery bears to a series of brain diseases. It is obvious that I desire and shall place the interests of the surgeon foremost in my observations. My individuality, or rather my convictions, however, will serve to limit the province of surgery rather than to extend it. Since the operation itself is regarded as devoid of danger, we are very apt, nowadays, to chisel open and to incise in order to find out whether there may not still be products of disease that can be removed, notwithstanding all deliberation and the uncertainty of diagnosis: in other words, we are tempted to risk without considering what the purpose and the result of such risk is. I do not believe that I will diminish the limits of surgery, if in the following studies, the conditions in which surgical interference results in good—in even the best—chances of success are first investigated, and if indorsement of the operation be then limited to such cases. Hence many cases are certainly omitted which by some lucky chance may have been fit for operation. But I would not like to leave to chance or blind luck what should be accomplished solely by critically acquired experience and strictly scientific examination. Even though hesitation lead to limitation, if the latter be wise, it is certainly the best road to wealth: understanding, care, and definite results are the lasting rewards. I hope for many and great results for surgery of the brain, even though at present I recommend only a small number and only selected cases as

coming within its scope. The success assured in such a limited sphere will be the agent of increased usefulness.

OPERATION IN CASES OF ENCEPHALOCELE.

A perusal of the more modern and of the older literature on hernia cerebri will show that the danger in the treatment of these pathological conditions is thought to depend upon the presence of the brain in the hernial sac. This explains the endeavors to obtain a positive differential diagnosis between meningocele and encephalocele; for in these days of antisepsis, interference is considered permissible in the former variety, as was advanced recenty by Sklifasowski (Sitzungsberichte der Moskauer chirurg. Gesellshaft, 1881, No. 13) and Flothmann ("Ein Beitrag zu den Operationen der Cephalocelen" in "Der Frauenarzt," 1887, No. 4), while in the latter form it is considered better not to attempt a radical cure (Heineke: "Die chirurgischen Krankheiten des Kopfes," 1882, S. 240).

There is certainly considerable difference in the various forms of cephalocele, according to contents, size, situation, and possibly development. The relations of the sincipital forms are the simplest and most constant. These emerge regularly through the ethmoid bone, causing a funnel-shaped depression in the cribriform plate; but, as can plainly be seen in the smaller examples of hernia, the opening into the funnel is formed laterally by the margins of the ethmoidal fissure of the frontal bone, while anteriorly and posteriorly a portion of the ethmoid bone is still preserved. This creates the impression that the disorder developed laterally at first and not in the median line. If the funnel-shaped opening be followed downward, we pass in three different directions in different cases: First, between the frontal and nasal bones, pretty direct from before backward to the nasal eminence; second, behind the frontal and nasal bones into the superior meatus, downward and laterally between the bone and the cartilaginous portions of the nose; third, into the orbits through a fissure in the suture between the ethmoid, frontal, and lachrymal bones. On this account, cephalocele sincipitalis has been divided into the naso-frontal, naso-ethmoidal, and naso-orbital varieties. As a rule, these tumors, communicating with the cranial cavity by means of the three canals just mentioned, are not pure meningoceles, but, to use the nomenclature of Heineke, are either hydrencephaloceles or cenencephaloceles. In both cases they

contain brain substance; in the former variety this is surrounded by a larger or smaller quantity of liquor cerebralis; in the latter, it is present without any surrounding cavity. The latter category is the one presented by the greater number of such tumors occurring on the front of the head. On this account, these are usually small tumors, with broad bases or only indistinctly pedunculated, feeling soft or at least not tense, pulsating in most cases, or becoming smaller during sleep and filling up during crying. In a large number, perhaps more than half of all cases that have been observed, the skin covering the tumor had become changed—sometimes becoming of a shining white and thin character resembling a delicate scar, at other times traversed by dilated blood-vessels, so that occasionally the entire surface would seem to consist of a telangiectatic angioma. This circumstance is important because it reminds us that in every case of telangiectasia of the nasal eminence or of the bridge of the nose, consisting of a projecting red mass resembling the cockscomb, we must not omit thinking of encephalocele.

For the pure or empty forms of encephalocele, which were formerly regarded in a *noli me tangere* manner, I would advise more active interference. Two years ago I first saw two cases of cephalocele containing brain substance, which had been extirpated by Schmitz, of St. Petersburg, with favorable results. Later, September 5th, 1887, a ten-day-old child was operated upon at my clinic for a swelling about the size of a walnut situated upon the left side of the nasal eminence. The surface of the swelling was red and presented an undoubted case of telangiectasia. Toward the cranium, the spheroid tumor was somewhat pointed so as to present a sort of pedicle which could be traced to the bony surface. In extirpating, a circular incision was made into the telangiectatic skin, and the thin pedicle which passed into the cavity of the cranium was tied with catgut. The swelling was found to consist of a piece of the substance of the brain about the size of a hazel-nut in which ganglion cells could easily be demonstrated. The cavernous tissue rested immediately upon this. The small wound, which was closed by the thread, healed rapidy and without any disturbance in the condition of the child. From the swelling a flat ridge ran to a cleft formed partly by the soft parts and partly by the bone; this cleft seemed to be between the

right nasal and the frontal process of the superior maxillary bone and extended into that side of the nose. The upper, narrow fissure spread out into a triangular opening, the base of which was directed toward the margin of the cartilaginous portion of the nose, and seemed to lie between the alar cartilage and the fibrous basis of the wing of the nose. Six months after the first operation, development having progressed satisfactorily so that the child seemed strong and healthy, this opening was closed by freshening the margins and transplanting the neighboring skin. At the present time, the child has progressed nicely, is able to stand and walk, and there is no trace of hydrocephalus nor of intellectual disturbance.

In cases of sincipital encephalocele, especially the smaller forms which are apt to be examples of cenencephaloceles, I would recommend the operation just described, which is rendered justifiable by our fortunate experiences. It is well known that children born with this malformation live longer than those afflicted with occipital cerebral hernia; in several cases they have been known to pass the age of puberty. At least, Larger[1] reported ten cases of adults with sincipital encephalocele, nine of which had preserved their intelligence. However, the majority of cases of this malformation die during the first days of life or at least during the first years; and even those which survive, do not escape secondary results of the deformity. Thus in Volkmann's[2] observation in a girl three and a half years old, the eyes were turned and pushed outward. The patient of Muhr's[3] was affected more seriously; for although he reached his forty-second year, he had been an epileptic since childhood and later was afflicted with epileptic mental derangement and progressive insanity.

Sometimes the increasing size and tension of the contents of the swelling cause a rupture of the overlying soft parts; sometimes such openings are caused by external injuries to which the cerebral hernia is especially liable on account of its position; such injuries may be penetrating wounds, or only bruises, causing sloughing and inflammation of the thin covering of the skin. The method of formation of the encephalocele explains, in part at least, why blows, pressure, bruises, and friction are so apt to destroy the purpose of these tumors; the latter are not only apt to be richly supplied with an anastomosis of blood-vessels, but are also often the seat of telan-

giectatic angiomata, as in my case; or the covering of skin is as delicate and fine as a thin, veil-like scar. In a work by my assistant, Dr. de Ruyter, which is about to be published, attention is called to the fact that the peculiarity of absence of the dura mater at the summit of the tumors of spina bifida, noticed by von Recklinghausen, also existed in several of our cases of occipital encephalocele. The soft, succulent, and porous structure of cavernoma undoubtedly blends with the layers next to the covering of skin—the pia mater and the arachnoid. Hence the serious import of an infiltration of blood and of the formation of a slough; hence also the necessity for operation.

This relation of the integument of encephaloceles also furnishes points regarding the etiology of the disturbance; and what we know regarding this is calculated to justify our recommendation of the operation of total extirpation. I am aware that the most important points in the history of the development of cephaloceles are still open questions. A glance at one of the most recent descriptions of this (Ahlfeld's) renders this plain enough. The etiology of these malformations is not explained by a single cause, but is ascribed to different circumstances; they are included, in part, in the chapter of malformations through amniotic obliterations, in part with hydrocephalus internus and externus as fœtal disturbances due to primary, general, or partial accumulation of fluid in the ventricles or membranes of the brain. As far as the scope of these pages is concerned, it is immaterial which factor we consider responsible for a sincipital encephalocele. Let us accept the standard description of Virchow ("Die krankhaften Geschwülste," Bd. I., S. 183). Under certain circumstances, as when the communications between the different ventricles are obstructed, the general distention in so-called hydrocephalus internus which affects the lateral ventricles especially, but which may also attack the third and fourth ventricles, may influence one of these cavities more than another. Virchow considers the obliteration of certain parts of the roof of the ventricles as quite common in his experience. A considerable collection of fluid may form behind one of these deformities, resembling a closed sac. A portion of a ventricle distended in this way and surrounded by a mass of brain is pushed outward and gives rise to this form of cerebral hernia, called hydrencepha-

locele. Naturally, it must be taken for granted that an opening in the skull either existed at the time when pressure was exerted outward, or was formed after a while as a consequence of this same pressure. I have not been able to find a single case of hydrencephalocele in which the supposed hydrops ventriculi saccatus of Virchow has been demonstrated; but one thing has been found often enough, and that is a free communication between the cavity in the hernial sac and one of the lateral ventricles, and the dilatation of the latter accompanied usually by that of its fellow.

If, differing from this idea, we regard the dropsical collection of fluid in the anterior horn of the lateral ventricle and also the general dilatation of the ventricles of the brain in hydrocephalus internus as more secondary than primary factors, that is, as effects rather than causes of the disturbance, the very frequent co-existence of sincipital encephaloceles and oblique facial cleft must serve to credit the cephalic flexure of the amnion with a greater share in the etiology than we have hitherto been disposed to give it. Morian's[1] studies of the oblique facial cleft have shown to what extent of frequency and in which sense amniotic bands are operative. In 34 cases of oblique facial cleft, anomalies of the brain were present in 26; among the latter number there were 7 cases of encephalocele with and without hydrocephalus, and 15 cases in which amniotic adhesions were still visible or demonstrable. Morian obtained interesting points in explanation of the manner in which amniotic bands may act. Until the time of Delsarte's well-known experiments, we considered only adhesions with the margin of the cleft; but Morian demonstrated that the most significant malformations of the cranium and skull could ensue through simple stenosis in the cephalic flexure. In this way, the amnion also becomes responsible without previous inflammations producing adhesions and obliterations entirely by delayed separation and loosening, in which case no visible trace on the skin of the fœtus need remain. It is not necessary to assume that an amniotic band capable of amputating a finger or even a fully-developed extremity need cut deeply into the flesh or bones of the developed face in order to cause a fissure; we need only assume that a band catches anywhere in the location of the forming head, or that a part becomes adherent for too long a time and thus

suffers from delayed separation, in order to understand how any pressure or traction, wherever it happens to be applied, may prevent the junction of the several pieces which together form the upper jaw at one of the sutures in question. Morian advances these possibilities in an elaborate manner for his second cleft—the one probably concerned in the child upon whom we operated. It is scarcely creditable that the frequent coincidence of the oblique facial cleft and cerebral herniæ of the fore part of the head can have any other than a common etiology. As early as 1792, Klein attempted to establish an etiological connection between a case of encephalocele which he described, and co-existing facial cleft. Be this as it may, whether we imagine the cephalic flexure of the amnion adherent at one point of what is to become the brain, or whether we ascribe the dilatation to a circumscribed increase of pressure in the contents of the brain vesicle, in either case, whether traction or pressure, the part concerned remains the same, namely, that division of the brain vesicle which afterward forms the anterior horn of the lateral ventricle. I have already called attention to the fact that sincipital encephaloceles are uniformly placed laterally. It is only in cases of large and extensive openings in the skull that the original lateral development is obscured and the impression of medial situation is created. Just as in large scrotal herniæ we cannot say whether the case is one of external or of internal inguinal hernia. In the case of Muhr (l. c.), the tumor seemed to be absolutely in the median line; but anatomical investigation proved that there were two adjacent herniæ: the frontal lobes of both cerebral hemispheres were elongated so as to form two prolongations which were enveloped by dura mater, but were separated from each other by the attachment of the falx to the crista galli. These cerebral prolongations consisted of gray cortex on their exterior, but in their interior did not present any extension of the lateral ventricle of the same side.

As in this case of double-sided pure encephalocele, the contents of the herniæ always show a development from either hemisphere only—from the anterior horn of either lateral ventricle. This fact decides the character of the brain substance lying in front; this is part of the frontal extremity of the affected hemisphere, a region of which a large part or almost all may be lost without causing any interference with any of

the functions of the brain. Therefore we can remove this projecting part of the brain, even when it has some size; we need not fear the loss of a motion or sensation. Hence, according to Virchow's investigations ("Geschwülste," Bd. III., S. 270 ff.), this extra-cranial mass of brain substance, if there be no co-existing effusion into the ventricles, really represents superfluous substance—a sort of heterotopic exuberance. If, in addition, we consider that perhaps it never had a place in the interior of the skull, but was developed outside originally, our cenencephaloceles would more correctly be regarded as small exencephalies—as "exencephalic protuberances." Etiologically considered, cenencephalocele and hydrencephalocele certainly belong together, not only because they emerge from the same parts of the skull, but also because a series of gradation may be observed between the cases in which the cavity of the lateral ventricle is continued directly into the interior of the encephalocele and those in which only a very fine canal leads from the ventricle to the part of the brain lying in front; and finally, even this connection is lost. In the last case only a solid ectopic mass of brain presents itself.

Therefore I consider it justifiable to treat cases of sincipital encephalocele by means of the radical operation of extirpation of the entire sac placed in front, including its contents.

The operation in cases of cenencephalocele is attended with a better prognosis than that of hydrencephalocele; not that there is any decidedly greater danger, but because the latter is complicated with an advancing hydrocephalus internus upon which, unfortunately, the operation does not seem to exert any influence. I cannot say with certainty that hydrops ventriculi is always absent in cenencephaloceles. Virchow's statements ("Krankhafte Geschwülste," Bd. III., S. 272) are decidedly opposed to such a view, for besides mentioning cases in which the dilated cavity of a lateral ventricle extended into the projecting portion of brain, others are reported in which the latter was entirely solid and yet a hydropsical ventricle was found at a certain depth in the cavity of the cranium. In certain herniæ of the occipital region both of these conditions existed together; through one opening in the vertical plate of the occipital bone there projected the posterior lobe of one cerebral hemisphere with a dilated and elongated posterior horn of the lateral ventricle; through the other, a solid mass,

with hydrops ventriculi more deeply, presented itself. This much, however, seems certain: that an effusion into the ventricles is often, perhaps even very often, absent in cases of cephaloceles which are filled with solid brain substance. Since, up to the present, extirpation of the projecting walls of brain substance has been resorted to very seldom and really only in cases of mistaken diagnosis, I am unable to add much to our knowledge regarding the subsequent absence of hydrocephalus. In this matter, I am influenced by the impressions of my own observations, in which there was no development, or more properly speaking advance, in the hydrocephalus, while in other cases of hydrencephalocele of the occipital region, operated upon by me with most favorable results, the hydrocephalus not only remained the same, but in the course of the following year it increased considerably. Holmes[5] reports a case of swelling at the inner canthus of the orbit in a young woman. Although it resembled an encephalocele, it was regarded as an atheroma, because the patient insisted that she had only had it three years and was positive it had not existed previous to this. It was only during the operation that the communication with the cavity of the cranium was discovered, after the portion of the brain contained in the swelling had been removed and unmistakably recognized by macroscopical and microscopical examination. Healing followed rapidly without any immediate or subsequent cerebral disturbance. Subsequently, Prescott Hewett also reported a successful removal of an encephalocele which had been diagnosticated as a simple cyst.

Whether the cases recorded by Larger in which persons affected with cerebral herniæ had grown up and remained healthy and free from hydrocephalus, had only cenencephalocele or also hydrencephalocele, I have been unable to ascertain. If my assumption that cenencephaloceles are not complicated or are less frequently associated with advancing hydrocephalus be correct, the operation of total extirpation would find its proper indication and would have the best chances in these tumors. While the subsequent hydrocephalus might furnish an important contra-indication against any operative procedure in cases of hydrencephale, this objection does not apply to cases of cenencephalocele, and hence the latter are the best cases for the operation.

Another result which might be accomplished by the operation is the prevention of the extension of hydrocephalus which already existed; this applies also to the operation for hydrencephalocele. A favorable influence upon the hydrocephalus through the operation would not be outside the range of possibility. Hence the justifiable limits of applicability of the operation might be extended so as to embrace this category of cephaloceles. I was careful not to omit mentioning this point, since in a series of cases, hydrencephalocele and cenencephalocele can only be distinguished from each other after the operation.

Among the individual cases of occipital encephalocele there exists a much greater variety than in the sincipital forms. The largest cerebral herniæ which have been observed belonged to the occipital region. These were cases in which the greater part of the brain was contained in the tumor, a sac having the size of the head being attached to a microcephalic skull with flattened roof; naturally these have no surgical interest. Similarly those cases in which the occipital encephalocele is merely part of a general rachitis need not be considered as regards question of operative relief—cases in which there is a separation of the laminæ of the upper cervical vertebræ, with an elongation of the foramen magnum and a prolongation of the latter far into the supra-occipital bone. The evident symmetrical form of the defect indicates a resemblance to the marked forms of spina bifida in which, as in these cases, the median line divides the opening or cleft into two equal portions. Independent of the size of the sac, the existence of the emerging nerves will prevent its removal. In a few cases even the fourth ventricle has been found in the sac; probably, in every case, it had become lengthened so much by traction that the nerves which are given off just behind the pons and from the lateral fissures of the elongated spinal cord, as well as the fourth ventricle itself, were exposed to the danger of injury during the operation.

Except in pure meningoceles which are actually found in this situation, the question of operation can only come up in cases of encephaloceles of the supra-occipital bone which are placed laterally. Most of these emerge above the external occipital protuberance, hence also above the tentorium. The opening in the bone through which they pass then not infrequently communicates with the posterior fontanelle.

It is not improbable that these forms of cephaloceles occipitales superiores are developed similarly to the sincipital forms. In support of this view may be mentioned the frequent absence of the dura mater from the summit of the swelling and the peculiar disposition of the pia mater, both of which peculiarities Dr. de Ruyter found existing frequently in our preparations of both the sincipital and occipital forms—a peculiarity which is quite analogous to the relations of the pia and the arachnoid in the tumor-like cases of spina bifida, as pointed out by von Recklinghausen. In this connection, the occurrence of two occipital encephaloceles next to each other in one of our cases, one associated with a separation of the laminæ of the upper cervical vertebræ, the other placed laterally in an opening in the supra-occipital bone, is of interest. I do not wish to go any deeper into this analogy, but will merely refer to the work of De Ruyter.

Just as sincipital encephaloceles contain the frontal extremity, the occipital have for their contents the occipital extremity of the corresponding hemisphere of the brain. But in the latter forms the cenencephaloceles are the rare examples and the hydrencephaloceles by far the most frequent varieties of these cerebral herniæ. Like the frontal extremity, the occipital end of the cerebral hemisphere may be lost without appreciable disturbance. Hence, there would be no objection to the removal of this portion.

After looking up a large number of casuistic papers and my own observations in quite a number of cases, I believe myself justified in asserting that, excepting in cases of pure meningoceles, cephaloceles occipitales inferiores represent the irregular and difficult varieties, while cephaloceles occipitales superiores are the more typical cases, like the sincipital examples. At the surgical congress last year, I presented a case in which a tumor of this region, larger than a walnut, had been successfully operated upon by me. Unfortunately hydrocephalus, which had probably already existed at the time of the operation, became prominent soon afterward, and since then has become larger and has produced more disturbance. The case which Alberti presented at the same session of the congress had a similar course in this respect.

CHAPTER II.

THE OPENING OF DEEP ABSCESSES OF THE BRAIN.

The disease of the brain in which there is no question regarding the value of surgical interference, is the formation of pus in the interior of the cerebral hemispheres—cerebral abscess. As long as the letting out of pus in general is a proper and indisputable task allotted to surgical skill, the attempt to evacuate pus from the interior of the cranium must be regarded as a laudable effort. Whoever agrees with us that abscess of the brain has no other course except a rapidly fatal bursting into the ventricle or an equally constant fatal diffuse meningitis, must regard the opening of abscesses of the brain as a rescue from urgent danger to life. Notwithstanding this, the necessity for trephining in cases of abscess of the brain has recently been doubted, for instance, by Rose.[6] These doubts are chiefly founded upon two circumstances: first, an anatomical fact—encapsulation of abscesses of the brain, and, second, upon the difficulty met with clinically of diagnosticating them. The influence of the former has been greatly exaggerated; that of the latter is constantly being overcome more and more.

Only abscesses which form acutely are free from encapsulation—*i.e.*, sclerosis of their surroundings. As is well known, these abscesses are found regularly upon the surface of the brain and most often as a complication of diffuse meningitis of the convexity. They occupy the situations of those portions of the cortex of the brain which have become bruised through traumatism: moderately large collections of pus beneath pia mater which has become infiltrated with pus. This cavity of the surface, filled with brownish pus, appears eroded and is surrounded by a layer of red softening, which again is enveloped by a yellow serous zone of œdema of the brain, the latter often being quite extensive.

The slowly developing chronic abscesses of the brain which really constitute the subject of our consideration are most frequently encapsulated. This capsule, which resembles a membrane, sometimes like a mucous membrane, is extraordinarily smooth on its inner surface; and it can also easily be separated from the surrounding parts of the brain, from which it separates the abscesses. This dense wall, which is often quite thick, has erroneously been considered not only as a limit between abscess and brain, but also as a protection to the brain against the spread of the collection of pus. But the formation of the encapsulating wall is an indication that the process of suppuration has come to a standstill, and that the entire process has become devoid of danger as far as the latter is dependent upon the progressive destruction (breaking down) of the substance of the brain. However, every abscess of the brain which causes fatal consequences through rupture into the ventricle is apt to be surrounded by a membrane. Since rupture is due to a constant increase in the size of the abscess, there can be no doubt that even the encapsulated abscess of the brain enlarges regularly and constantly at the expense of the neighboring white and gray matter. In this way, anatomical investigation shows quite often a continuity between the membrane and its immediate surroundings in a condition of the white softening which precedes suppuration. It is contrary to all experience and observation to regard the encapsulation of abscesses of the brain as a process of limitation which initiates retrogression—a process of healing. The case reported by Rose as supporting this view is easily explained in another manner. A man, aged twenty, who had had an otitis complicated by severe but indefinite brain symptoms while a child, died of pneumonia. Post-mortem examination showed from five to seven abscesses in the white substance, some of them surrounded by a membrane of the thickness of the back of the blade of a knife, and with these was also caries of the petrous portion of the temporal bone. It is not at all necessary to associate the occurrence of brain symptoms which appear during the course of ear affections—as was the case ten or fifteen years previous in the example cited—with the formation of one of these seven abscesses. The latter were developed, unnoticed and insidiously, as usual, during the course of a chronic aural and bone affection, as shown at the autopsy;

the absence of capsule is not, therefore, any proof of age or even of retrograde changes.

There is so little hope that a collection of pus in the brain, when not interfered with, may find a lucky escape through the ethmoidal plate into the superior meatus of the nose, or through an opening in the roof of the tympanum into and through the ear, that we cannot count upon it. With the exception of a few cases in which pus escaped from the depths of the brain through an opening produced by a fracture, the envelopes of the brain having previously become adherent one to another and to the bone, all histories of cases which would seem to support the occurrence of such perforations may be regarded as errors or unfounded assertions of physicians. Rose's case was an instance of this: at the autopsy the opening in the dura mater and in the ethmoidal plate could not be found, but the place of rupture into the ventricles was discovered. The old case of Santesson ("Hygiea," 1862, Bd. 14), in which post-mortem examination demonstrated a communication between the cerebral abscess and a longitudinal fracture of the Eustachian tube, has but a single similar modern instance in the case recorded by Barr,[7] which also proved fatal. The latter case was under the care of MacLeod and presented an abscess in the frontal lobe due to traumatic necrosis of the skull. Pus had found its way along the base of the skull to the petrous portion of the temporal bone, had broken through the roof of the external auditory canal and escaped through the ear. In the very best cases, these uncommon means of escape have been narrow and unfavorably situated.

What was formerly believed regarding deep cerebral abscesses is still true. If the knife of the surgeon is not applied in time, death will be the only result. The only objection which can be urged against trephining for the removal of intra-cranial collections of pus is the difficulty in diagnosis. But these difficulties are not as great nor as insurmountable as they seemed formerly. Under certain circumstances the diagnosis can be made with great certainty. Therefore it is our duty to become acquainted with these circumstances more closely and to understand them.

The etiology is of the greatest importance in the diagnosis of abscesses of the brain. This will be readily understood when we remember that there are no idiopathic or primary

abscesses of the brain; every cerebral abscess is of deuteropathic origin—*i.e.,* has originated from a distinctly different disturbance which is associated with suppuration. Abscess of the brain is either associated with traumatism which has produced an open wound in the soft or hard walls of the cranium, or has arisen from some other form of suppurative process of the skull—most often suppuration in the ear. All other abscesses of the brain are either metastatic or tuberculous.

The older medical writers considered idiopathic abscess of the brain as of quite common occurrence; more modern authors either say nothing about it or emphasize its rarity, or, like Goldstein,[8] are sufficiently critical not to allow an unexplained case to serve as basis for a crumbling doctrine.

Goldstein's case occurred in a butcher, thirty-three years old, who was seriously ill when he presented himself for treatment. Five years previously patient had had gastric fever lasting two weeks. At a later period he suffered from a short attack of melancholia with religious hallucinations. Three years after this he had a lump on the right side of the neck; this was said to have disappeared after inunction with blue ointment. At this time he began to be subject to headaches, which at first occurred at least once a week. Vomiting had not occurred at any time. In the course of the succeeding year, neuralgia of the fifth nerve followed. Finally (January 7th, 1888) symptoms indicating pressure upon the brain, vertigo, and slowing of the pulse appeared. On January 20th, the consulting physicians found: Sensorium free, speech dragging, slow, no form of aphasia. Excruciating pain on right side of cranium; this is increased by pressure upon certain points, especially the parietal region above the ear. Left arm somewhat paretic, left leg more so. Knee-jerks, right stronger than left. Sensibility normal. No fever. Respirations normal. Frequency of pulse, 40 to 32. Bilateral choked disk; dilated pupil, right side. Several hours afterward the pareses of left arm and leg had increased to paralyses, and the sensorium began to become clouded. January 21st.—Complete coma; no answers to questions. Trephining over region of greatest tenderness by Prof. Riedel. A piece of bone measuring almost 4 cm. was removed from the cranium by chisel and mallet. A greenish-yellow, purulent mass representing broken-down brain substance infiltrated with pus,

flowed through the opening after division of the bluish and very tense dura. The adjacent parts of the brain were removed with the knife, and the opening into the cranium was enlarged so as to measure 5 cm. in diameter; as a result of this, additional brain substance of a normal appearance was pressed forward. (Microscopic examination revealed pus-corpuscles, broken-down brain substance, and giant cells; hence the diagnosis of cerebral abscess.)

Immediatly after the operation, the frequency of the pulse was increased to 72 and the sensorium again became clear. After nine days, patient could stand and walk. The choked disks also disappeared. But a large, pulsating prolapse had formed at the site of operation. The improvements in general condition and in motor disturbances continued during the succeeding months. However, the prolapse increased in size, gave rise to considerable discharge, and presented gangrenous degeneration. During April, spasms of the left angle of the mouth set in and the patient again began to complain of vertigo and headache: later there were added spasms of the left arm and leg. May 7th fever and rapid diminution of strength were observed. Additional pareses of the left side of the body. Ineffectual attempts to relieve by removal of the cerebral prolapse, which was permeated with collections of pus. Death, May 13th, four months after trephining. The autopsy revealed meningitis and also the left middle lobe changed into a whitish-yellow mass. "The posterior border of the fissure of Sylvius is soft and forms the anterior boundary of a typical abscess of about the size of an apple surrounded by a pyogenic membrane; communication of this with the larger abscess could no longer be demonstrated with certainty. The right lateral ventricle presented a rupture into its roof." I have repeated these data from the history of the patient because they establish two points—the position of the chronic abscess was established from the first when the dura was incised, it being found immediately beneath this membrane; and the great infiltration of the projecting mass of brain substance with pus—hence only an infiltration and no free pus. The author himself says: "The only point which the previous history of the patient gives us is the occurrence of a lump in the neck in the course of the previous year. The discovery of a second abscess at the autopsy perhaps supports the supposition that

there existed metastatic products having their origin in the carbuncle (?), since multiplicity in such cases is the rule. But we cannot assert this with certainty. The details of the occurrence of the second abscess still remain somewhat clouded." I consider myself also justified in regarding this case as dark and unexplained.

In the majority of cases of metastatic abscesses of the brain, these are but part of the manifestations of pyæmia, and therefore are multiple, corresponding to the well-known collections of pus in the lungs, liver, kidneys and joints. I once saw over a hundred abscesses in all sections of the brain of a man who died from gangrene of both legs produced by frost. On the other hand, there are some few cases in which the metastatic abscess of the brain remains single—the only occurrence of suppuration widely separated and distantly removed from the original collection of pus. These are found in certain lung troubles and pleuritic affections. After Virchow had described metastases in the brain in cases of gangrene of the lung, Biermer directed the attention of practitioners to metastatic suppuration in the brain following bronchiectasia. A comprehensive review based upon eight cases of his own observation, has recently been published by Näther.[9] This is a report of patients who had suffered from severe inflammatory affections of the respiratory organs—abscess of the lung or gangrene of the lung following infarction, pneumonia, or injury to the lungs, also pleurisy with effusions and empyema. These patients suffered from a distressing cough with exacerbations, accompanied by a periodical or constant expectoration of a fœtid discharge rich in pus. There was fever due to acute exacerbations having the symptoms of pneumonic infiltration and lasting a variable length of time. This condition lasted years or even tens of years, the patients constantly becoming more cyanotic and cachectic and constantly suffering more from dyspnœa, and finally dying from general debility or presenting the symptoms of a serious affection of the brain. In almost one hundred cases of gangrene of the lung, fetid bronchitis and bronchiectasis examined in the dead-house of Leipzig, Näther found collections of pus in the brain exclusively, in no less than eight cases; the other organs, though altered as a consequence of the lengthy course, presented no abscesses. Seven out of these eight cases of ab-

scesses were multiple, being found in various parts of the brain; in one case only was the abscess single. In still later communications, attention is again called to the multiplicity of abscesses occurring under these circumstances; for example, Hein ("Bericht über die Rudolph-Stiftung in Wien," 1883) found no less than seven abscesses in the frontal lobes, corpus striatum, and lenticular capsule in a case of broncho-pneumonia from foreign bodies. On account of this multiplicity there is at present little prospect of successful operation in these cases. However, if with one of the affections of the lung or an empyema even during the stage of absorption, the symptoms of a large abscesss of the brain showed themselves, the previous or existing disease of the lung would be an important aid to the diagnosis. Such a case seems to be presented by the masterly observation of Senator:[10] A stone cutter, aged thirty, was admitted to the hospital, presenting dulness over the right upper lobe, offensive expectoration, and evening elevation of temperature. Then, without any other brain symptoms, there followed a paralysis of the upper extremity which gradually increased and extended from the fingers to the arms. Five days after the commencement of the paralysis, patient was seized with an epileptic attack which began with spasms of the right hand and then of the right arm, while still conscious. On the eighth day a paresis of the lower branches of the right facial nerve was observed, and on the ninth day also a paresis of the right leg, again most marked in the foot. During the increase of the paralyses a disturbance of speech was noticed, and on the thirteenth day this developed into a well-marked ataxic aphasia. The patient died on the seventeenth day. The autopsy revealed several cavities and numerous peribronchial zones; in the interior of the left hemisphere an abscess containing 50 gm. of fetid pus, not surrounded by a distinct membrane and showing a tendency to rupture toward the periphery. In this situation a visible flattening and greenish discoloration extending in front of and along the fissure of Rolando across the frontal lobe. The posterior portion of the third frontal convolution was especially involved in this process.

Under certain circumstances, as in the case just related, it would be possible to diagnose the seat of the disease of the brain, while the coexistence of the affection of the lung would

have served to differentiate the existence of an abscess from the presence of a tumor.

Consideration of this sort induced Drummond [11] to undertake an operation which, though unsuccessful, demonstrated the existence of a single abscess in the neighborhood of the opening made by the trephine.

A delicate anæmic woman, aged twenty-nine, a laundress by occupation, was taken sick two weeks before her admission (December 18th, 1886), with repeated chills. On admission, she complained of dyspnœa and pain in the right side of the chest, in which an extensive pleuritic exudation was easily demonstrable. Two days later she had a chill with subsequent dulness of intellect and signs of a broncho-pneumonia over the left lung. She became very weak and miserable as a result of persistent vomiting and diarrhœal discharges. There was some improvement in her condition from December 26th to January 4th, when there was another chill and a return of the vomiting and diarrhœa and later symptoms of a fresh pneumonia, lasting until January 10th. Exploratory puncture having revealed empyema, this was evacuated through an incision beneath the angle of the scapula and the pleural cavity was drained and washed out. The offensive odor of the pus which existed at first disappeared and the secretion became less in amount. February 11th there was another chill and signs of a pneumonic infiltration in the apex of the left lung; this disappeared in the course of a week. February 18th the drainage-tube was removed, and the patient progressed favorably during the subsequent ten days. February 28th there was another chill, but this was unaccompanied by any disturbance of the pulmonary organs. March 3d, three convulsions, which could not be described any more fully; these were repeated on the 7th and 8th. She complained of violent headache in the left parietal region. Ophthalmoscopic examination showed optic neuritis of both sides. March 9th, convulsions of considerable duration, which were observed fully. They began with twitching of the right upper lid, followed by conjugate deviation of the eyes toward the right and immediately thereafter a turning of the head toward the right; then followed other spasms of the alæ of the nose and of the right angle of the mouth, also rigidity and spasms in the right arm and leg; then the left leg and the left arm also became rigid

and affected with spasms as well as the left side of the face. Between the different attacks there was an interval of two to three hours, during which the turning of the eyes and head still continued. After ten repetitions the attacks became constant, and there was developed a paralysis of the right leg and arm, the left extremities remaining rigid; the patient became aphasic. Since the temperature was elevated, an abscess in the region of the left motor area was diagnosed and an attempt was made to discover this abscess by trephining. After the most careful determination of the position of the fissure of Rolando, an opening $2\frac{1}{4}'' \times 2\frac{3}{4}''$ was made over the lower portion of this sulcus. Despite repeated punctures with the exploring needle after incision of the dura, a collection of pus could not be found. During these proceedings considerable cerebro-spinal fluid had escaped; probably on this account there was an improvement in the pressure symptoms. The next day there was full consciousness, speech had returned, the convulsions had ceased, and the paralyses had disappeared. But a high fever and a delirium, in which the patient got up and ran about, set in. Nine days after the operation, the patient died. At the autopsy it was demonstrated that the opening made by the trephine corresponded to the lower third of the ascending frontal convolution and the adjacent portion of the ascending parietal convolution (the anterior central) and in front reached also to the lower fourth of the inferior frontal convolution. But the abscess was found in the lower end of the middle frontal convolution; the pus was thick and green and the abscess about the size of a hazel-nut surrounded by an œdematous zone and separated from the pia only by a thin layer of gray substance.

Larger collections of pus in cases of tuberculosis of the brain, resembling the traumatic forms, have only been observed twice. Once in the highly interesting case of C. Wericke,[12] in which Hahn opened the skull, incised the brain, and evacuated the abscess; and again in A. Fränkel's case.[13] Purulent degeneration of larger conglomerations of tubercles of the brain usually affects the centre only, while the periphery remains of the well-known dry, cheesy mass. Even the softened product resembles thickened milk rather than pus. In the two cases of Wernicke and Fränkel it was different. The collection of purulent material resembled an ordinary abscess

of the brain, except that the layer of granulations of the encapsulating membrane of the abscess was filled with tubercle bacilli and that the pus was rich in these micro-organisms. Fränkel's case is certainly of especial pathological value because it demonstrates what it would have been necessary to examine by those who imagine having observed an idiopathic, non-tuberculous abscess of the brain.

I do not believe that we can hope for much in opening tuberculous abscesses of the brain. It must be difficult to scrape out the cheesy infiltration in the soft structure of the brain. What was left unremoved would cause an extension of the breaking-down process until it had arrived at the ventricle and perforated into it, which would result in the usual final termination—death. We shall encounter the consideration of tuberculous affections of the brain again in the chapter on the operative treatment of tumors of the brain. It will suffice to state here that the occurrence of primary and exclusive tuberculosis of the brain, at least in cases in which the question of the existence of a tuberculous abscess of the brain presents itself, is more than doubtful.

At present we can only count upon successful operation in cases of cerebral abscesses due to traumatism or to aural and other forms of suppuration of bone. Hence the importance of an early and certain diagnosis in these cases.

Let us begin by considering the first variety—abscesses of the brain due to a wound of the skull or its soft spots. In the consideration of these, we exclude acute abscesses of the cortex which occur directly beneath the point of fracture at the situation of the injury to the brain; the latter represent the retention of pus in wound cavities or beneath a fragment of bone. If the disturbance in the process of healing, the characteristics of the bottom, margins, and surroundings of the wound, the altered appearance of the granulations, and the escape of pus between the fractured particles do not call our attention to the imminent danger of the patient, the picture of a developing diffuse meningitis will soon disclose the fact that it is too late for us to interfere.

As a matter of fact, these abscesses can seldom be distinguished from traumatic, purulent meningitis of the convexity. The picture[14] of the latter, with its excitation and paralytic symptoms, coincides with that of abscess of the cortex. It is

comprehensible, therefore, that the diagnosis of the latter has been based more upon the changes in the healing wound than upon its special symptoms. However, keeping in mind three recent highly successful cases of trephining, it will be well to call attention to a couple of diagnostic points. Suppurative meningitis is usually developed early and rapidly, and generally so violently that in the shortest time the function of that part of the cortex first involved is interfered with. On the other hand, the development of abscess of the surface requires a certain length of time; it can scarcely be expected before the second week after the injury. If development be even slower and the appearance of the abscess delayed, the surrounding membranes of the brain may have become matted together so firmly and strongly that a sort of barrier is formed about the collection of pus. This is the condition under which such an abscess may exist without extension of the meningitis and may be successfully evacuated. Briefly, then, the late appearance of so-called meningitic symptoms—one or two weeks or later after the injury—the cessation or markedly protracted course of such symptoms, and finally the prominence of the paralytic symptoms, may, in a given case, call attention to circumscribed abscess of the brain and hence to a condition amenable to surgical therapeutics.

Esmarch [15] treated a case occurring in a man twenty-eight years old who had received a punctured fracture of the left parietal bone; five days afterward he felt a weakness of the right foot which extended upward and finally involved all the muscles of the right side of the body. The next day there were very painful spasms in the regions of these muscles and of the right side of the face and spasmodic contraction of the muscles of the neck. On the seventh day difficulty of speech and vomiting. Complete motor paralysis of the right side of the body; retained consciousness and reflex excitability. During the succeeding three days he had a daily attack of spasms in the paralyzed muscles. Thick speech. Loss of consciousness. At the first medical examination on the sixth day after the injury, fluctuation was obtained around the wound, which was covered with a scab, and at the margin a loose splinter of bone could be felt. Trephining on the eleventh day after the injury. The wound was 1.5 cm. long, semilunar in shape, and bounded by discolored, granulating margins. Two small

splinters of bone were removed and the opening in the bone was enlarged with the chisel and bone-forceps. During the efforts to control severe hemorrhage from the dura, a fair quantity of yellow, creamy pus, devoid of odor, escaped. Three days after the operation, patient awoke from his soporific condition and regained full consciousness. The muscles regained their power in the reverse direction in which they had become paralyzed. The patient recovered completely.

I admit that this case could easily have been taken for one of diffuse meningitis, and that the poor condition of the wound at the time patient entered the hospital was the only thing that would have induced me to trephine. The cessation of symptoms during the three days preceding the operation is the point wherein this case differed in its symptoms from the ordinary ones of meningitis of the convexity. However, the opening of the skull and the improvement of the condition of the wound would be indicated in such a case of meningitis of the convexity, which otherwise would be entirely hopeless if a bad-smelling or inflamed wound were found over the seat of a splintered fracture.

The behavior of the wound also served as a basis of the diagnosis of abscess in the following case: Elcan [16] reports the case of a boy, five years old, who had a complicated fracture of the frontal bone, through which opening brain substance presented itself. After several days, coma, aphasia, and hemiplegia. Laying bare of the fracture and elevation of a number of fragments of bone. During the following days an extensive prolapse of the brain occurred, hemiplegia and aphasia became complete, but consciousness returned and remained intact. The seat of fracture was again exposed four days after the first operation, since the fragments of bone had again become dislocated and seemed to be separated. In removing the fragments of bone a large abscess was opened into and six to eight ounces of pus were evacuated. The recurrence of cerebral prolapse was prevented by a pressure bandage. The paralysis and aphasia disappeared after a while. Eighteen months after the accident the boy had recovered completely, both mentally and bodily.

In this category also belong the cases of Mason [17] (recovery after trephining in the second week) and Mosetig [18] (Heilung nach Ausmeisselung der durch Schuss gebrochenen Knochenstelle in der 3. Woche).

There is a marked difference between the occurrence of these early abscesses forming at the end of the first and during the second week after the injury, often simultaneously with the development of a suppurative leptomeningitis, and the formation of those collections of pus in the brain which occur later and do not make their presence known until weeks, months, and even years. The former originate in the bruised portions on the surface of the brain, the irritation causing inflammation and suppuration arising from the wound of the skin and the spaces in the shattered portion of the skull. The contused area is to a certain extent converted into a collection of pus, there is a transformation of the red softening—the thick, reddish mass representing the bruised portion of the brain—into a purulent infiltration and subsequently into a cortical abscess. It is different with the deep abscesses situated in the interior of the hemispheres; it is certain, *a priori*, that they do not originate in the contused portion of the brain, since the bruises and lacerated wounds which the brain receives are practically always found on the surface of the organ, sometimes directly beneath the seat of injury, at other times diametrically opposite to this point. The late abscesses are placed beneath the cortex. It is only exceptionally, in cases of severe forces which have shattered the entire skull, that bruised areas have been found in the interior and even in the nuclei of the brain. But these cases of severe forces breaking or crushing the entire skull either cause death immediately or they heal without suppuration; hence they do not lead to the formation of abscesses of the brain. From what has just been said, it is evident that when we find abscesses of the brain occurring late and encapsulated, beneath the cortex within the white substance, they are not developed from pre-existing contused areas, or at least not usually. Their method of occurrence certainly does not differ from that of the non-traumatic abscesses of the brain associated with bone or aural suppuration, and like these they represent not continuous but disconnected suppuration. Between the suppurating wound or the bony ulceration and the abscess is a layer of brain substance which is apparently healthy. How the irritation to purulent degeneration is carried into the depths of the brain—whether the veins or the lymphatics surrounding the vessels are the routes carrying this to one or more of their nutrition territo-

ries—will not be discussed here. We will only consider here the different methods and states of development of acute and chronic abscesses of the brain of those having received injuries to the head, since such variations influence the symptoms and hence also the clinical characters of the abscesses which we are discussing.

It would be important for the diagnosis of traumatic abscesses of the brain if the method of the preceding injury offered any special peculiarities. But in this respect only one fact is certain and forms a general basis in connection with the injury: the solution of continuity of the surface. No abscess will result if the outer surface of the skull, the integument, remain unbroken, whether the bones be fractured or the substance of the brain be bruised or lacerated. This is, however, the only constant peculiarity of those injuries to the head which may result in abscesses of the brain. In cases of subcutaneous or simple fractures, whether longitudinal, multiple, or depressed, if the integument be entire, an abscess of the brain never results. This is not a deduction from the present theory of the formation of pus, nor a deduction from the assumption that every case of suppuration is due to infection from without through the tissues; it is the simple result of experience. No case has yet been published or described, or mentioned in any other way, in which abscess of the brain had resulted from a subcutaneous injury. The few cases belonging to a past age, which are enumerated conscientiously in monographs, are examples of most indefinite observations or are arbitrarily brought into this category. Among them is a history in which it is stated that a fistulous tract led from the exterior into the skull! In other cases no cognizance was taken of the extension of the apparently subcutaneous fissure in the bone—its connection with air-conducting spaces, through the middle ear, the cribriform plate of the ethmoid bone or the windings of the frontal sinuses.

The fact remains: Every traumatic abscess of the brain must have been preceded by a wound which communicated with the surface in some way, whether directly with the surface of the skull or indirecty through the cavities and passages connected with the ear and nose.

It is scarcely possible to give any further information concerning the peculiarities of a wound which predisposes to ab-

scess of the brain. Gun-shot wounds in which the projectile or parts thereof remain in the brain, punctured wounds in which the broken-off point of the knife remains in the cranial cavity, and comminuted fractures of small extent in which the broken bone consists of fine particles which are driven deep into the brain, seem to be followed more frequently than other injuries, by abscesses of the brain. The forms of fracture which are readily and often followed by tedious suppuration, supported by the fact of the occurrence of cerebral abscesses with traumatic necrosis of the skull, leads us to believe that long-continued suppuration is much more apt to be responsible for these late abscesses than the retention of foreign bodies and fragments of bone. However, it is only true in a general way that long-continued suppuration of the external wound may be regarded as a predisposing factor in the production of deep abscesses of the brain, since quite a number of abscesses occur after comparatively rapid closure of the external wound. Nevertheless, we may regard the endeavors of the present period to improve the form of complicated fractures of the skull when they are such that rapid healing is interfered with, as much a prophylaxis against the development of cerebral abscesses as a means of bettering the disinfection of the wound which the universally employed chiselling and cutting away the fracture particles afford. We hope that our present method of treating recent perforating, comminuted, and depressed fractures, as well as the injured brain, will result in a constant diminution in the frequency of late abscesses of the brain.

Finally, we must also remember that it is not essential that an open wound of the bone or brain exists; even a wound of the soft parts may be sufficient to produce an abscess of the brain. How this impulse extends through the bone into the depth of the brain has not yet been sufficiently investigated; but there is no doubt regarding the occurrence of abscess under these circumstances. The greater number of such injuries are forms of suppuration, and usually these have a history of lengthy duration. Sometimes they were limited to the skin and the subcutaneous tissue so that the bone did not seem to be involved at any time, but at other times they induced osteitis and necrosis, which again led to the formation of pus, and thus an extension of the suppurative process occurred.

The latter variety of cases leads us to the consideration of those abscesses of the brain which result from the development of a non-traumatic, suppurative process in the bone or soft parts of the skull. These comprise, in the first place, diseases of the middle ear. Probably one-half of all cases of abscesses of the brain are induced by the ear. Though no figures are at hand to estimate this, a considerable number of fatal cases of abscess of the brain occur in which neither the physician nor the family of the patient knew that the latter had suffered from trouble with the ear; only at the autopsy was the caries of the temporal bone revealed. In still a greater number of cases the aural affection is probably overlooked at the post-mortem examination, and especially was this the case in former years during which little or nothing was known concerning the affections of the middle ear. At that time an abscess induced from the ear was regarded as an idiopathic one.

The nature of the affection of the ear decides the diagnosis of abscess of the brain. Almost always the chronic affections of the ear are the ones which precede, and usually have preceded a long time, the formation of a collection of pus in the brain. It is imporatnt to know this in making the diagnosis of abscess of the brain in patients affected wtih aural disease. The diagnosis is dependent upon the history of chronicity of the previous suppuration. Even acute exacerbations of chronic otorrhœa have no influence upon the tendency to or the growth of abscesses of the brain; the added symptoms due to such exacerbations must not be regarded as manifestations of the supposed brain affection, even though they consist of vertigo, headache, drowsiness, and numbness, but rather as symptoms of an increase in the severity of the disease of the ear. The form of chronic suppuration which concerns us in this connection is not that of the outer ear nor originally that of the internal ear, but suppuration in the middle ear, the result of otitis media chronica purulenta. In the great majority of cases the disease dates back to childhood, sometimes beginning as an acute inflammation, at other times commencing chronically. The etiological influence of scarlatina, diphtheria, small-pox, typhus fever, and local tuberculosis of the nasopharyngeal cavity or of the temporal bone is well known. The seat of suppuration is the cavity of the tympanum with

its communicating pneumatic cavities, generally associated with purulent or ulcerative disease of the bone, less frequently without such bony disease.

Severe brain symptoms occurring during the height of an acute inflammation of the middle ear, even though non-suppurative, have often been described. Vertigo, high fever, clouded intellect, even convulsions and other deceptive symptoms, such as dilatation of the pupils, ptosis and parases of the legs in such cases, have disappeared immediately after paracentesis of the tympanic membrane.[19] We may assume that all such disturbances occurred in consequence of an increase of the intra-labyrinth pressure, due to the distention of the middle ear with pus.

The possibility of an acute purulent inflammation of the interior of the ear causing abscess has been pointed out through observations in a case of purulent meningitis following a simple, non-suppurative catarrh of the middle ear, published by Zaufal and Schwartze.[20] Through the kindness of chief staff surgeon Schmidt, of Metz, I add the following interesting history of a case of abscess following acute suppurative otitis media, which has been published in the mean while in the "Archiv für klinische Chirurgie."

A man of twenty-two who had never before had any aural trouble, was affected with an otitis media of the left side since several days; the symptoms were so moderate that no paracentesis of the drum-membrane was performed. In the course of the first week, pus found a spontaneous opening, without, however, any consequent amelioration in the inflammatory symptoms; suppuration became quite profuse, but did not involve the mastoid process. After daily washing out of the middle ear through a catheter the symptoms subsided. In the fourth week of the patient's illness, during the night of July 2d, he was seized with severe pains in the occipital region and repeated vomiting. Temperature, A.M., 102°; P.M., 103.5°. On the afternoon of July 3d, paresis of the left facial appeared. There was an improvement in the following days, so that by July 8th the headaches and the purulent discharge had disappeared and there were scarcely traces of paresis. During the following three weeks there was no return of the discharge, but the patient complained of severe headache of

the left side, vomited a number of times, and was constipated. Twice a slowing of the pulse to 48 was noted. There was no elevation of temperature. The fundus of the eye remained normal. The voice in a whisper could be heard at four metres by the left ear, which now appeared healthy, the defect in the membrane no longer being visible. In the seventh week there was a repetition of the severe pains in the head in the left parietal region, but these were only occasional. The sensorium seemed affected, patient not answering questions directed to him correctly and replying slowly. He felt weak. Vomiting once. No fever. No slowing of the pulse. The skull, which until now had not been sensitive to percussion, became tender over the left frontal region; this did not extend to the temporal region. Later, with a continuance of the amnesic disturbances, general hyperæsthesia set in. The weakness gradually increased. At no time was there fever or slowing of the pulse. September 7th, a diminution in the power of motion of the right upper extremity, as compared with the left, was observed. September 8th, somnolence. September 9th, restlessness, attempts to get up, sudden death. The autopsy revealed an abscess in the left temporal lobe, about the size of a hen's egg, separated from the surface by a thickness of 1 cm. of creamy, soft cortical layer and enveloped by a strong membrane 1 mm. thick. Next to this abscess, which extended from in front to the first temporal convolution, there was a second one; the latter was also closed, of about the size of a walnut, and extended up to the neighborhood of the posterior horn of the lateral ventricle. The pus of both abscesses was thick and devoid of odor. There were no thrombi in the sinuses. Lungs normal. No other collections of pus in the body. The examination of the temporal bone after removal showed normal relations—drum-membrane delicate and entire, ossicles and their attachments intact, middle and internal ear and mastoid cells free from changes, no trace of pus anywhere. The auditory and the facial nerves were unchanged.

This very interesting case would certainly have been regarded as an instance of idiopathic cerebral abscess, if the otitis media had been overlooked during life. As it is, this observation coming from an experienced aurist and the absence of any involvement of the lungs or other parts of the body as

shown by the autopsy, show that even after an acute suppurative otitis media, an abscess of the brain may ensue in the typical position—the interior white portion of the temporal lobe of the same side. The report is all the more important because, as the autopsy showed, at no time had there been any extension of the affection to the mastoid process or the periosteum of the auditory canal.

Schmidt enhances the value of his observations by adding an analogous case of J. Gruber,[21] in which, however, the extension of the suppuration to the periosteum and the cells of the mastoid process had led to a previous oncotomy. A laborer, aged forty, was affected with a severe inflammation of the middle ear of the right side, with perforation of the drum-membrane. Soon after periostitis of the mastoid region with the formation of abscess was added. The abscess was opened and the disease was cured in six weeks; the patient heard a watch at the distance of 6 cm. Three months after his discharge, he returned complaining of severe headache; on this account he was admitted to the medical clinic; his illness was diagnosed as gastritis. The headache increased during the following days and high fever was added. On the third day there was loss of consciousness and on the fifth day he died. The autopsy revealed the existence of an abscess in the right temporal lobe, about the size of a hen's egg, the walls of which indicated that it had existed for some time. Not the slightest anomaly of the temporal bone could be found, with the exception of a moderate thickening of the drum-membrane and of the lining membrane of the mastoid cells.

The cases of Macewen and Horsley cited below also show that subacute and acute suppurative processes of the ear may cause abscesses in the temporal lobe.

Only a minority of the abscesses of the brain in question lie immediately next to the diseased middle ear, so as to be, in one sense, continuous with the suppuration in the interior of the bone. In these cases an opening in the roof of the tympanum has also been found, and a direct contact of the dura mater, thickened or permeated by small collections of pus, with the suppuration within the middle ear. In this way it may happen that the dura becomes separated from the bone to a greater or lesser extent by the pus. E. Hoffmann[22] has recently compiled such cases under the name of pachymenin-

gitis externa purulenta, occurring after inflammations of the middle ear, and has discussed them quite deeply according to their pathological and clinical value. A typical abscess was found in the substance of the brain over the spot at which pus had been found between the dura and bone. It looks as though the continued existence of a collection of pus between the bone and dura could cause and favor the subsequent occurrence of cerebral abscess.

As an example, an autopsy is cited, made in Schwartze's clinic, the case being one in a man who died from pyæmia. The cause of the pyæmia had been a crumbling thrombus in the transverse sinus. The roof of the tympanum presented a perforation about the size of a pea. The dura over this spot was lifted up and the space thus produced was filled with fetid greenish pus. Greenish, greasy pus is likewise evacuated from the brain corresponding to this situation, and is seen to proceed from a ragged-walled cavity in the temporal lobe about the size of a pigeon's egg.

The collection of pus between dura and bone is sometimes quite considerable, and may become more formidable through extension along the surface. As a rule, it extends along the veins and thus reaches larger or smaller branches of the veins.

Bezold[23] reports the following autopsy: The dura mater over the roof of the tympanum was lifted up and infiltrated by pus over an area of 2 × 1 cm. A similar infiltration extended along the petrosal sinus up to its junction with the transverse sinus. The walls of both sinuses, especially the latter, were infiltrated with pus. Beneath the elevated dura, the tegmen appeared discolored, presented a number of red points and several small superficial defects in the bone, one of which, about the size of a pinhead, permitted the passage of a probe into the middle ear. The tympanum and the mastoid antrum were filled with a dirty yellow, creamy mass.

The cases in which the pus spreads from the posterior surface of the pyramid of the temporal bone to the posterior occipital fossa are of special importance in this connection. The position of the thick, greenish, and fetid pus covering the surface of the bone and lifting away the dura, sometimes extends far in all directions, at other times only following the transverse sinus in the interior of the cranial cavity.

Field[24] found at post-mortem examination in a man of

twenty, who had been subject to intermittent attacks of otorrhœa since childhood, and who had a collection of pus in the mastoid cells, that the pus had found its way through the thin walls of the sulcus for the lateral sinus without affecting the latter, and had pushed forward between dura and bone to the region of the left parietal bone; in this situation a considerable collection of pus was found.

An extension of pus along the transverse sinus into the inferior posterior cranial fossæ seems to precede abscesses of the cerebellum with special frequency. In these cases there is usually also thrombosis in the sinus which has become softened and surrounded by the pus; however, there are plenty of cases in which the intravenous blood remains fluid. Thrombosis of the sinus behaves differently. Sometimes it remains limited to a short distance, as for example when confined to the segment contained in the sigmoid groove; sometimes it extends into other sinuses or into the cerebellar veins, or descends through the lacerated foramen into the internal jugular vein. The thrombus itself also indulges in as many variations. It may become soft and brittle and thus be the source of embolic abscesses of the lung, or despite the suppuration it may become hard and dense, leading to the formation of connective-tissue plugs which obliterate the sinus. Cases have been described in which the thrombus was found organized for a part of its course, and beyond soft, puriform masses were found. Perhaps this is of some importance in explaining the discontinuity of many deep abscesses of the brain from the point of original suppuration. It is especially in these cases that the route by which the excitant to suppuration is carried into the depths of the brain has been investigated but very little.

In a case of tuberculous osteitis involving the first two cervical vertebræ and the basilar portion of the occipital bone, I found at the autopsy an obliteration of the transverse sinus, also of the superior petrosal sinus of one side, without any visible disturbance in the middle or internal ear. In the occipital and temporal lobes of this side there were, however, small abscesses. Is it not plausible to suppose that the abscesses had formed around thrombi extending along the venæ cerebri inferiores which had become organized in both sinuses, but had degenerated in the venous branches which emptied into

the sinuses? Around the broken-down thrombus the periphlebitic intracerebral abscesses formed. This supposition possesses more probability in explaining the unknown method of occurrence of the abscesses in question than the extension of the inflammation along the lymphatic perivascular spaces. After the abscess has become formed, traces of the method by which its generators have reached the depth can occasionally be found in the obliteration of the veins just mentioned.

At any rate, the majority of cases of abscesses developed in the course of an otitis media are removed from the region of primary suppuration, and found in the interior of the white substance separated from the ear by layers of unchanged brain substance.

Barr[25] reports 76 cases of abscess of the brain following aural suppuration, 7 of which he observed himself; in 55 cases the abscess was situated in the temporal lobe, in 13 cases in the cerebellum, in 4 cases in both the cerebrum and the cerebellum, in 2 cases in the pons, and in 1 case in the crus cerebri. In all cases the abscess was in the same side as the affected ear. It is not stated how many of these cases were encapsulated nor how often meningitis and thrombosis of the sinuses coexisted. One interesting peculiarity, is however, mentioned: the fetid character of the pus in 69 out of the 76 cases.

Körner[26] gives a tabular *résumé* of 67 cases of cerebral abscess due to otitis. Thirteen of these were associated with thrombosis of the lateral sinus, 31 were situated in the cerebrum, 19 in the cerebellum, 4 in both the cerebrum and the cerebellum. Another fact of importance is shown by Körner's deductions: the greater frequency of abscess of the brain on the right as compared to the left side, a relationship which is preserved in all intracranial affections following diseases of the middle ear and caries of the mastoid. Among 151 cases, 90 were on the right side and followed an aural disease of the right side, and 57 were on the left side following an affection of the left ear; 4 cases were bilateral. Since disease of the right middle ear is not more frequent than of the left, Körner attempts to explain the fact that the meninges, vessels, and brain are more apt to become involved after suppurative processes of the right ear, by the anatomical differences in the sigmoid fossa of the two sides. On the right side this fossa extends

further in front and externally in the base of the pyramid than on the left side, in 77 per cent of the skulls which have been examined.[27] Furthermore, on the right side, the bony wall which separates the collection of pus from the cavity of the cranium is usually thinner than on the left side, and therefore favors the extension or the rupture of the suppuration. In doubtful cases this might induce us to diagnosticate an abscess of the brain with more probability when on the right side than on the left.

The study of this *résumé* shows that when the petrous portion of the temporal bone is affected more on its surface, the abscesses will occur in the temporal lobes; but when the suppuration occurs in the mastoid cells, the posterior fossæ and the cerebellar hemispheres are the parts which are apt to become affected. But since otitis media suppurativa usually affects both the middle ear and the interior of the mastoid process, these facts can only be utilized in distinguishing a few isolated cases. Most often it is even impossible to tell which of the temporal bones is the more affected. Barr demonstrated five skulls in cases of abscesses in the temporal lobe, in which the tegmen tympani was destroyed by ulceration, and mentions that 27 cases of caries of the petrous portion of the temporal bone are noted among the cases which he compiled.

As a matter of fact, the discovery of a carious process of the walls of the tympanum by means of careful probing strengthens a presumptive diagnosis of abscess, since deep cerebral abscesses have been found more frequently after cases of otitis complicated by osteitis and caries than after those cases which are not accompanied by degeneration and suppuration of the bone.

All that Hoffmann states regarding the relations of pachymeningitis suppurativa to aural suppuration is also applicable in this connection. The occurrence of a collection of pus between the dura mater and the bone, as well as of cerebral abscess, is favored by a perforation in the drum-membrane which is situated unfavorably or is too small. Suppurations in the upper part of the tympanum are especially serious in this respect; first, because this portion may become separated from other cavities in such a manner that the products of inflammation stagnate, and, second, because the bony roof sepa-

rating the pent-up masses from the dura is formed of a lamella of bone of special thinness in this situation. Stenoses and obstructions in the external canal, interfering with the ready escape of pus, act in the same manner—for instance, the obstruction due to granuloma, the polyp of aurists. Finally, it may be mentioned that cholesteatoma of the petrous portion of the temporal bone has, in more than one case, not only given rise to absorption of the bone, but also to an abscess in the temporal lobe.

The intracranial diseases which complicate otitis media suppurativa are, besides deep cerebral abscess, the separation of the dura (just mentioned), leptomeningitis purulenta, and thrombosis of the sinuses. In any given case we would have to differentiate between these three affections.

In those abscesses of the brain which occur with ear troubles and which we are called upon to diagnose, an important factor connected with their development is their insidious beginning. This latency from the beginning and often lasting a relatively long time causes the greatest difficulty in the diagnosis; but it also offers the possibility of distinguishing them from cases of meningitis and phlebitis with their decided and sudden advent.

Contrary to what has just been stated, it is very largely believed that concussion, often quite insignificant, or injury to the head accompanying aural trouble, is the cause of the abscess. This idea cannot be correct, because in cases in which death followed immediately or shortly after the traumatism which was charged with the result, a large abscess was always found. However, that the incriminated traumatism eventually causes a rupture into the posterior horn of the lateral ventricle or an increase in the growth of the abscess, is rendered plausible in those cases that are followed by an immediate change for the worse in the general condition of the patient or even by a rapidly fatal result. I have had two occasions to observe in the practice of aurists that trephining the mastoid process was followed by a rapid change in the abscess from its latent condition to a fatal termination.

Suppurative processes occurring with or without osteitis in other portions of the cranium are less significant in the history of cerebral abscesses, as compared to the importance of aural suppurations. In two cases of perforating tuberculosis

of the cranium, I have found abscesses beneath the diseased portion of the occipital bone, in the occipital lobes. In the first case, which was fatal, the two deeply placed and encapsulated abscesses, contained thick, greenish pus, which led me to believe that they had been produced by the suppurating process of the bone and not by softened tubercles. In my monograph on injuries to the head ("Kopfverletzungen," S. 510), I have mentioned cases of abscesses of the brain after caries of the ethmoid occurring with an ulceration in the nose and after old cases of caries of the frontal bone. Since then several similar observations have been published. It has not come to my knowledge that one of these cases was successfully trephined. Since most of the abscesses of this sort are situated in the frontal lobes, it is comprehensible that they were overlooked during life and were not discovered until at the time of the autopsy.

The symptoms of every abscess of the brain, whether due to traumatism, otitis media, or any other form of suppuration in or about the skull, are divisible into three groups. First, those which are due to the suppuration itself. These will resemble the disturbances which every deep-seated collection of pus produces. Second, symptoms indicating an increase in intracranial pressure and a disturbance and change in intracranial relations. These will correspond very much with those of cerebral tumors. The third group comprises symptoms due to the particular seat of the abscess. It is quite evident why special stress should be laid upon the last group of symptoms.

Were there no other suppuration except that which forms the abscess of the brain, the symptoms of the first group, and especially the fever, will be of the greatest value for our diagnosis. I shall illustrate this in an example cited below. Let us suppose a case in which a complicated fracture of the skull has been received, which heals in a short time after an unimportant or scarcely perceptible amount of suppuration, but which caused an abscess in the brain, say in the frontal lobe. Three months later this man, who had previously been in good health, begins to complain of all sorts of disagreeable sensations—coldness of the fingers, chilliness, despondency, and loss of appetite, these symptoms being especially marked at night. The measure of temperature shows an evening rise, this fever being usually low; it may last days and weeks and

may then disappear. Then it will return after some time, and then will be more intense than upon its first appearance. This periodic appearance undoubtedly corresponds to the successive increase in size of the chronic abscess. Accordingly the intervals during which there is no fever may last for months and even years. In this manner the fever may be repeated several times. If we succeed in observing these nocturnal fever movements, their irregularity, their variations, their intermissions and remissions, we will have a very important point directing our attention to the formation of an abscess of the brain otherwise latent, providing that the traumatism has become healed and the wound no longer suppurates. The shivering and elevation of temperature are of no value whatever in the diagnosis of cerebral abscess when associated with suppurative otitis, for besides the suppuration in the brain which we are trying to discover, there exists another—that in the ear—which is apt to give rise to these same symptoms with each retention of the pus in the mastoid cells or anywhere else in the interior of the ear. The same applies to the other disturbances in the general health of the patient due to the pus and the fever: the weakness and indifference of the patient during the periods of fever, which form a contrast with the relative freshness in the early morning, also the disturbances of digestion, such as gastric pressure, distended abdomen, nausea, and tendency to vomiting terminating in efforts at vomiting and in actual vomiting.

Generally there will next be added to the symptoms enumerated as due to the suppuration, the first evidence of an increase in intracranial tension, seldom absent—headache. In many cases nothing more can be said about these headaches except that they are long-continued and variable in intensity and that they have one constant peculiarity: their regular exacerbation during the existence of fever; even though they have been absent or insignificant during the absence of fever, they always increase so as to become especially severe and intense when the patient's fever returns. Everything which increases the blood-pressure in the cavity of the cranium, through congestion or stasis, such as excitation through use of alcoholic drinks, bending over and a low position of the head, will give rise to those pains or increase them. In some cases, especially in those situated in the temporal lobes and

cerebellum occurring with otitis media, the headache is fixed, strongly localized, and corresponds pretty accurately to the situation of the abscess. It increases or occurs immediately upon percussing the affected part of the cranium. In such cases we should, therefore, never fail to percuss. As a matter of course, tenderness upon pressure over the pneumatic cells of the temporal bone is only a sign of inflammation or of inflammatory products in its interior. In the same way tenderness beyond these limits, but still in the immediate neighborhood of the ear, as for example 2 cm. behind the external auditory canal, would point more properly to a collection of pus between the dura and bone than to an intracranial collection. In cases of aural suppuration and suspicion of an abscess in the temporal lobe, this symptom becomes important only when the region of the squamous portion of the temporal bone and of the posterior inferior angle of the parietal bone is sensitive to our percussion.

The remaining symptoms of pressure are rarely as marked in cerebral abscess, excepting in the last stages, as they are in cerebral tumor. These are also distinguished by periodicity and variation in intensity. It arouses suspicion if, during the evening fever and headache, the pulse-rate diminishes and the drowsiness of the patient becomes marked. In one of my cases of abscess of the cerebellum, I have observed periods of the most severe pressure symptoms—unconsciousness, coma, slowing of the pulse to 30, stertorous breathing, and Cheyne-Stokes' phenomena. After some time all these serious disturbances had disappeared. A period in every minute of which the worst could be feared gave way to one of marked improvement. This changing is more likely to occur and more often in the picture of cerebral abscess than in that of tumor of the brain, which is distinguished especially by the continuity and steady increase in the pressure symptoms. In both cases the symptoms become worse after a time; in abscess, this is spasmodic; in tumor it is more regular. In addition also, in the course of time, the choked disc in the fundus of the eye is not apt to be absent, while in abscess this symptom, which is so important for the diagnosis of tumor, is noted only in a small fraction of the cases.

We now come to the third group of symptoms of an abscess of the brain—that of the area symptoms, which are given

a deciding place in the diagnosis. As a rule, they are of great importance when the abscess is situated in the neighborhood of the motor area, but of little value when it is situated in the frontal, occipital, or temporal lobes.

The number of cases in which traumatic abscesses develop beneath the parietal bone is, considering the frequency of localized fractures in this region, not a small one; so that unmistakable area symptoms have often aided in making a diagnosis.

The area symptoms of an abscess of the brain are dependent upon the destruction of brain substance, or at least upon the softening of the latter which surrounds the collection of pus in its periodic increase in size. There may be a retrogression, in which case one or more of the area symptoms dependent upon the abscess will disappear. The collection of pus in the interior white matter of the brain at first simply separates the tracts without interfering with them. If the gray matter remains intact, the abscess may assume great dimensions, so as to involve an entire cerebral lobe or even an entire hemisphere without producing any area symptoms. The nearer the abscess approaches the cortex, however, and the more the brain substance is destroyed, the sooner may we count upon the appearance of area symptoms.

In connection with the therapeutic significance of the evacuation of abscesses of the brain, it is important to distinguish between exclusion symptoms which result from destruction of brain substance and those which are merely due to the temporary inflammatory and œdematous imbibition. The latter will disappear after the evacuation of the pus and the resulting diminution in the tension of the tissues and liberation of the circulation, while we cannot expect this from the former class of symptoms. In this way we explain the cure of paralyses, as for example is mentioned in the case which is described below.

So much has been written and discussed regarding the symptoms of localization of brain diseases in the cerebral hemispheres, that I will not need to enter into particulars concerning the area symptoms and their very important combinations here. In the examples of successful operation of cerebral abscesses we will, however, notice them.

As early as 1854, Belmonte, of Göttingen, at Baum's sug-

gestion, compiled the successful cases of evacuation of abscesses of the brain. Blum gives a report of 44 cases of trephining in the 19th volume of Langenbeck's "Archiv;" these were all performed for cerebral abscess, and half of them recovered. Seydel[28] collected 13 cases of trephining of cerebral abscesses, treated antiseptically, during the past year, with 6 recoveries. The examples which I add will be restricted to cures during recent years, except in my own cases.

Rivington[29] describes a very instructing case: A man of thirty-one was injured by a file in such a manner as to cause a penetrating wound of the soft parts at the postero-superior angle of the right parietal bone. The bone did not seem to be injured and the patient did not seem to realize the severity of the accident. Three days later there were headaches, which constantly became more severe; several days later, delirium and left-sided hemiplegia were added. On the 27th day trephining was performed. Incision into the dura resulted in the evacuation of considerable pus. The pains ceased at once, and recovery resulted soon, so that after 31 days the patient could resume his occupation of letter-carrier. Nine months later (December, 1876) mild spasms occurred, which increased rapidly in intensity and frequency and terminated in paralysis of the left side. Reopening of the old wound resulted in the evacuation of some serum and coagulated blood. Soon after patient was able to raise and lower his left arm. Consequently it was supposed that the scar was the cause of the attack. As early as the following February, 1877, the patient performed all his duties at the post-office, and this continued for over two years, until July 18th, 1879, when the convulsion came on again, having been preceded for several days by headaches. Since the convulsions continued to become more severe, the reopening of the scar was again resorted to, but without success this time; the arm and the leg remained paralyzed and painful and speech became thick. Convulsions were not repeated, though otherwise the condition remained the same, with increase of the diminution in sensibility and mental weakness. In October patient was admitted to the London Hospital; at this time he could not walk unaided, dragged the left leg, and could not move the left arm with the exception of the fingers, which he could open and close. There was also paresis of the lower branches of the left facial.

Speech unintelligible. Great restlessness in bed, so that he required a separate nurse. He answered questions correctly, but when left to himself he became delirious and gave evidences of illusions regarding himself and his surroundings. Optic neuritis of both eyes. Rivington found a slight swelling of the scar, suspected abscess of the brain, and opened the scar. He found beneath a necrotic portion of bone and removed this. During the following night the wound opened and a large quantity of pus escaped. The patient began to improve immediately in every way; he regained the power to walk; speech became more intelligible; he could move the left arm and fingers freely. The improvement was lasting; even the remaining weakness of the left extremities diminished in the course of time. The swelling and opacity of the optic discs diminished and the acuteness of vision improved. During November of the same year a transient retention of pus caused another relapse with headaches and a convulsion which commenced in the fingers of the left hand and rapidly became general; after this the left side was again paralyzed for some time. This was the only disturbance prior to the closure of the wound in December. Patient was discharged in the best of health on January 8th, 1880.

This case illustrates the peculiarities of a paralysis originating in the brain and especially from the motor area. The leg is less paralyzed than the arm, and this condition of the arm is incomplete since the fingers can still be moved. The lower branches of the left facial were paretic, but not the upper, for the patient could close his eyes. Furthermore, pronunciation was interfered with, speech became thick and indistinct. All these disturbances are initiated by an epileptiform convulsion which is extraordinarily characteristic of the first area symptoms. Once, at least, it was proven that the convulsions proceeded from the paralyzed upper extremity. Preceding the area symptoms were the persistent headaches and the eventual development of choked disc. This case is also instructive in that the same convulsions and paralyses, preceded by severe headaches, occurred with each renewal of the collection of pus or interference with its escape. Finally, we may undoubtedly attribute the improvement after the first operation of opening the scar down to the dura, to resulting diminution in the intracranial pressure which had favored the occurrence of œdema in the periphery of the abscess.

Morini Vincenzo[30] began to treat a thirteen-year-old boy during the end of April, 1887; the boy had received a complicated fracture of the left parietal bone through a stone thrown at him; he had been unconscious for some time. Upon recovering consciousness, he was aphasic and had paralysis of the right side. The inability to speak disappeared after forty days, but the paralysis continued. The wound, which was treated with cold compresses, suppurated considerably and closed only after a time and incompletely, so that, upon admission, there was still a small space in the bifurcated scar measuring 7 × 4 cm. over the plainly depressed parietal bone, which space was filled with spongy granulations. The lower border of the depressed area was 3 cm. above the auricle. The granulations allowed a fine probe to enter the cranial cavity for a distance of 8 cm., and this resulted in the escape of a few drops of pus. There was incomplete paralysis of the extremities and of the facial nerve of the right side. The reflexes were exaggerated. Sensibility preserved. Two or three times a day there were tonic contractions of the paralyzed right arm, lasting a short time. In the beginning of May chiselling out of the depressed bone. The exposed dura was smooth and shining and did not pulsate. In the centre an opening was found which was undoubtedly a continuation of the visible loss of bony substance. Incision of dura and evacuation of over 40 grams of greenish pus. Irrigation of the deep abscess cavity with 1:1,000 sublimate solution, and scraping of its walls with the sharp spoon. With the exception of an elevation of temperature of 100.8° in the evening of the second day, there was an absence of fever and rapid healing. Cicatrization was complete by the end of July. The paralysis improved markedly. Patient was able to walk. The muscular power of the upper extremity also increased very much.

This case illustrates the importance of long-continued suppuration at the seat of a fracture in the production of an abscess, and that the escape of pus from an opening between the fragments and upon the deep introduction of the probe is a point in the diagnosis of abscesses. It is evident that the close approximation of the fracture fragments caused a retention of the products of inflammation, and after this a suppuration of the originally bruised portion was produced. The paralysis could not disappear, since it was due to the injury which caused destruction of brain substance.

Those symptoms of excitation and of exclusion which serve to diagnosticate the exact seat of the brain lesion, are peculiar to and are often found only in abscesses beneath the parietal bone, in the neighborhood of the motor area. Roberts[31] presented, at the American Surgical Association meeting, an extensive table determining the points at which the cranium should be opened, with each of the area symptoms which are observed in this connection. Numerous other works, among which that of Horsley[32] is probably the most elaborate, enable us to expose and to reach the fissure of Rolando and the neighboring convolutions with great certainty.

But even without developed area symptoms, the association with a bloody traumatism and the succession in which the symptoms about to be enumerated were developed, were sufficient to enable Fenger[33] to make the diagnosis in the following case with perfect certainty:

A Chicago policeman received a pistol-shot wound from a prisoner on December 3d, 1882. The ball (32 mm. calibre) penetrated the upper margin of the left orbit at its outer and middle third. The bone was fractured. A fragment of the bullet lay above and external to the entrance and was cut out. Antiseptic dressing. On the third day there was a high temperature, severe headache, vomiting once, considerable œdema of the upper eyelid, and moderate exophthalmus. The headaches increased in severity during the following two days, but no other brain symptoms were added. The fever became higher. The skin extending from the point of entrance to the comminutely fractured upper border of the orbit was divided freely. Between the bony fragments were found pieces of lead. The frontal sinus had become opened, but its posterior wall seemed intact. An abscess containing about two drachms of pus found an opening through the orbit. No fracture was found in the roof of the orbit. Pulse and temperature now became normal and headaches and œdema of the lids disappeared. Three weeks after the injury, the patient felt perfectly well, slept well, his appetite was good, and he went out. Now and then he complained of headaches which proceeded from the left frontal and occipital regions; these did not last longer than from two to four hours and were not accompanied by nausea or vomiting. In January he had recovered sufficiently to be able to resume his work. While in the police station on

January 5th, he fainted suddenly, was driven home in a carriage, and seized with violent vomiting and pain on the left side of the head. No elevation of temperature. Pulse 70. Great weakness and indisposition. The pulse-rate diminished to 54 by January 12th. The patient, who answered questions at first, constantly grew more somnolent and finally became deeply comatose. Temperature remained normal. On this day trephining upward from the supra-orbital margin toward the semicircular line. The underlying dura was of a normal color, but so tense as to bulge into the trephine opening. There was no visible pulsation of the brain. A crucial incision was made into the dura. There were no evidences of meningitic exudation. Exploratory puncture with a hypodermic needle, at first downward in the direction of the orbits; suction through this only brought blood and brain substance. Then the explorations with the needle were continued internally, behind and externally, followed by aspiration, but without success. A longer needle was now used. After the latter had been introduced about two and one-half inches in an inward and backward direction, it became filled with a thin, fluid, semi-transparent, but slightly fetid mass. The needle was allowed to remain in its position to serve as a guide. A closed dressing-forceps was introduced along this guide. After opening the extremities, an opaque fluid and then thick yellow pus escaped. The opening into the abscess was kept open by a drainage-tube. Drops of pus began to flow out immediately. Iodoform dressing. Three hours after the operation the patient regained consciousness and was able to answer questions. Pulse rose to 64. In changing the dressings the abscess cavity was washed out carefully and gently with a solution of boric acid and a new drainage-tube was inserted. Until January 30th the condition of the patient improved markedly. No fever, no headache. Sleep and appetite good. The drainage-tube was shortened each day. February 12th, after having been up for a short time, patient began to complain again of headache and nausea. A week later the pulse was slower, temperature slightly elevated, and the patient had again become stupid and somnolent. Another operation was undertaken February 20th since a retention of pus was diagnosed. Exploration and aspiration through a long needle were again resorted to. After several ineffectual attempts,

the abscess was finally reached in the same direction as before. The syringe was filled with a teaspoonful of yellow pus. A longer drainage-tube was inserted in the direction found by the exploration. There was subsequent improvement and diminution in the production of pus until April 10th, when the tube was removed. April 15th the patient got up, and May 15th he resumed his duties of police officer. He had no headaches and had regained his former strength. Memory, speech, and all other brain functions were in the best condition. Ptosis and slight strabismus of the left eye. More than a year later, in July, 1883, he had three epileptiform attacks, associated with headaches, in the left frontal region. December, 1883, had another epileptic attack. Last year, no additional attacks. The headaches, which at the beginning were present in the frontal region, gradually began to disappear. Patient did full duty, and complained of nothing except of being less robust and of being tired out more easily than formerly.

Gussenbauer's[34] observation is of the greatest value: On the 12th of June, 1883, a man of eighteen was wounded by the discharge of a 7 mm. revolver held close to the forehead; about 3 cm. above the supra-orbital margin and somewhat to the right of the median line, the discolored entrance to the canal of the ball could be seen. The patient was entirely conscious and there were no traces of brain symptoms. Upon examination and enlarging of the margins of the wound the flattened projectile was found against the apparently uninjured bone. Healing progressed without disturbance. No fever; but little secretion. On the eighth day there was a discharge of a necrotic piece of connective-tissue. On the evening of the tenth day, temperature 101.3°. Two days later he complained of headache on the left side and pain in the left ear and the left eye. The wound was again enlarged down to the white bone, which looked dead; this was done on account of retention of pus. From June 25th to July 3d, evening exacerbations of temperature to 103°. Since the patient had suffered from ear trouble in previous years, necessitating the opening of the mastoid process two years before, the occurrence of the fever was attributed to a recurrence of the ear trouble. After July 3d there was no fever: the outer table of the frontal bone exfoliated slowly, so that on the first of August the necrotic piece could be removed. During this time there

was no fever. During the middle of August — eight weeks after the accident—he began to complain of headaches and converging strabismus was noticed. Beginning of September, paresis of the left facial; this disappeared after ten days, but returned toward the end of the month. A diminution of the mental power was also noticed. Bodily temperature was normal; pulse slowed between 60 and 40 beats per minute. From time to time vomiting occurred; this usually lasted one or two days and was especially apt to occur if the patient attempted to sit up in bed. There was a tendency to constipation. Once there was involuntary micturition. October 10th, the patient was again admitted to the surgical clinic of Prague. He had become indifferent, weak and emaciated, and sat up in bed with the head flexed and bent toward the left side. Sensorium seemed clouded, so that it could not be ascertained whether he recognized his doctor. With weak voice he complained of headaches. When left to himself he became somnolent very easily. Ophthalmoscopic examination showed pretty marked choked disc on both sides. Pulse 56. The assumption of an abscess in the right frontal lobe led to immediate trephining. Corresponding to the spot at which the necrotic bone had been removed, a depressed scar was found. Commencing at this point, the preserved inner table was chiselled away and an opening of sufficient size was made with the use of Luer's chisel-forceps. Upon removing the bone over the greater falciform process, hemorrhage from the corresponding sinus occurred. After ineffectual attempts to stop the bleeding by means of the application of the thermo-cautery, it was controlled by digital compression, continued during the entire operation. Fluctuation could be felt in the depths through the dura and still plainer after this membrane had been incised. The pia mater was œdematous and the brain substance beneath was in a condition of red softening. Puncture 3 cm. deep in the substance of the brain. This being followed by the escape of pus, the puncture was enlarged by incision and stretching with the fingers; a quantity of thick greenish pus escaped. The abscess-cavity seemed to be as large as a moderate-sized apple. Abscess-cavity and wound were irrigated with 1:1,000 corrosive sublimate solution and a drainage-tube was inserted into the former. A compress of sublimated gauze was placed upon the wound in the sinus.

After the operation the patient recovered consciousness pretty rapidly, and the pulse increased from 46 beats per minute before the operation to 104. The paresis of the facial had disappeared by the evening of the day of operation. The next day it was again somewhat developed. There were more intense headaches, which, however, soon disappeared. Pulse, temperature, and respirations remained normal. October 17th, drainage-tube was removed. By this time, the strabismus had also diminished. The patient was improving visibly. In the beginning of November there was a relapse. Abnormal convergence of eyeballs, choked discs on both sides; the granulations became excessive and tumefied so as to overhang the margins of the wound. Exploration of the depths did not reveal any pus, but a sequestrum 2 cm. long was found and removed. After this, improvement again took place and the ocular manifestations also disappeared. Toward the end of November, cicatrization was complete. The patient continued well until April 13th, 1884; then he began to have fever at night and right-sided headaches and nystagmus-like movements of the eyeballs. April 18th, after this change in the patient had become apparent, he talked incoherently and looked absent-minded; then he had epileptic convulsions with unconsciousness, the spasms being limited to the distribution of the right facial and to the right upper extremity. After this, patient recovered completely so that he was able to resume his college studies. June 19th, he had another convulsion, again preceded by headaches; on the 21st he was again able to go to school. In October, patient had a mild attack of typhoid fever, upon recovering from which he was well until May 10th, 1885. On this day he began to complain of headaches in the right temporal and supra-orbital regions. May 18th, he had an epileptic attack; this was followed by another attack after fifteen minutes and by subsequent ones at short intervals. The convulsions began with a turning of the head toward the left and tonic spasm of the muscles on the left side of the body, followed by clonic spasms, which soon extended also to the right side. May 19th, status epilepticus, increasing cyanosis, collapse, and death.

The autopsy showed, besides hyperæmia and œdema of the brain, complete cicatricial closure of the abscess-cavity in the middle and superior frontal gyrus. Careful examination of

the brain could not discover any inflammatory process or an encapsulated cerebral abscess. It remains unexplained whether the cicatrix in the brain caused an irritation of the surrounding brain substance, which caused the epilepsy, or whether the closure of the cerebral cicatrix by the scalp was the cause of this irritation.

March 29th, 1886, a sixteen-year-old collegian, H., was admitted to my clinic. Several hours before, he had shot himself in the head with a 7 mm. revolver. Immediately after the shooting, there was a short period of unconsciousness, but no other brain symptoms. The point of entrance, the margins of which were blackened, was 5 cm. behind and 3 cm. above the outer angle of the right eye. Around this spot there were ecchymoses. The surroundings of the wound were shaved and disinfected; then an antiseptic dressing. During the first four days there was apathy and drowsiness, frequent vomiting, slowing of the pulse, and headache. The headaches lasted ten days; this was longer than the other symptoms. The wound having healed beneath the scab without reaction, and the patient feeling perfectly well and strong, he was discharged April 22d. July 10th of the same year his family physician brought him to the clinic again. Intense headaches, especially in the occiput, had become developed, also repeated vomiting, constipation, and slowing of the pulse to 50 per minute. Off and on there were twitchings of the right angle of the mouth, but no other motor or sensory disturbances. Ophthalmoscopic examination showed marked choked disc on the right side. July 12th, paresis of the left facial, especially its upper branches (orbicularis). Temperature of surface 38.2°, but 1.5 to 1.8° higher at the cicatrix than over other portions of the head, especially than at the same point over the left side of the head, while the right axillary cavity showed 0.4° less than the left.

I made the diagnosis of abscess in the left frontal lobe from the preceding history of injury. Infection and inflammation might have been caused by the bullet, which probably entered the skull, instead of by external suppuration, which was absent in this case. This diagnosis found additional support in the successive development of headaches, the tendency to vomiting, the slowing of the pulse, and the choked disc limited to the side injured by the bullet. All these symptoms depend

upon an increase in intracranial pressure. It is well known, as has been stated above, that choked disc is absent more frequently in cases of abscess than in cases of tumors. In many cases, however, as in the one just mentioned, it is plainly often markedly developed. This will not seem surprising when we remember what variations in pressure exist in different abscesses. Sometimes after opening it streams forth, at other times it merely runs out drop by drop. If under certain circumstances, as in the preceding case, the choked disc is present, we must regard it as a valuable symptom, especially when, as was also the case in the history mentioned above, it corresponds to the side involved by the traumatism.

I wish to dwell only upon one of the fever symptoms of our patient: the temperature distribution of the head. Upon the diseased side the temperature was 1.8° higher than upon the healthy side. As far as I know, Broca was the first to point out the existence of differences in temperature on the two sides of the head in diseases of the brain. These differences can be ascertained with ease and certainty by the use of v. Anrep's thermometer. More frequent measure of the temperature would have shown also the evening exacerbation: but as it was, we had the evidences of elevation of temperature in general.

When the frontal lobe is the seat of the abscess, there are no symptoms from among the third group which are of any value in making the diagnosis, since, as is well known, this part of the hemispheres may be almost entirely destroyed without giving evidences of this. The larger the abscess—*i.e.*, the nearer it approaches the posterior divisions of the frontal convolutions—the more likely are we to have strabismus, disorders of speech or facial irritation, and paralyses now and then. The facial paresis in my case seemed to indicate a relatively large abscess.

After proper preparation of the field of operation according to the rules which I laid down as early as 1880,[35] and more explicitly in 1886,[36] after shaving the surface and cleansing with soap and water, as well as with ether and sublimate solution, I made a T-shaped incision through the cicatrix. The fibres of the temporal muscle were drawn aside and the frontal bone exposed up to the temporal surface, near the upper margin of the orbit. After careful and complete control of hem-

orrhage a piece about the size of a dollar was removed with chisel and mallet. There was very little to be seen of a bony cicatrix; but on the external surface corresponding to the cicatrix there was an osteophytic collection. The dura, pressed into the defect, pulsated plainly but did not give the feeling of fluctuation. Even after removing it I did not get fluctuation. Repeated punctures with a hypodermic needle to a considerable depth were unsuccessful. I did not get pus! Since there was considerable hemorrhage from one spot between dura and bone, and since now and then there was bleeding from the blood-vessels of the pia mater on account of the injury in wiping and in puncturing, I covered the entire surface of the wound with iodoform gauze, and without attempting to close the wound in the skin, I placed upon this our usual antiseptic occlusive bandage. The condition of the patient remained the same during the following three days. Subsequently, in changing the dressing, on removing the iodoform gauze considerable pus escaped, probably from one of the punctures. I then at once proceeded to divide the brain substance which still covered the abscess, with a scalpel, and entered a large abscess cavity. The membrane which lined the latter was so dense and tough that I could grasp it with my fingers and with a dressing-forceps and could pull it out in one piece. Whether I also removed the bullet with this, I do not know, since it could not be found, as is explained below. The great loss of substance was filled up with extraordinary rapidity by a pressing forward of the brain. The wound was closed after introduction of a drainage-tube. Again, and this time also after three days, I was compelled to reopen it. The fever had not diminished after the operation; on the contrary, it rose to 104.6° on the evening of the opening of the abscess and remained high during the following two days. In changing the dressings the brain pressed forward, sponge-like, over the margins of the bone, just as in cases of prolapse; this led me to suspect retention of pus and to seek for the latter. This resulted in the discovery of a second abscess in close proximity to the first; upon opening this there was an abundant escape of pus. I also removed the encapsulating membrane, as in the case of the first abscess. After this the fever diminished, but patient remained in a somnolent condition. There was no repetition of the prolapse; on the contrary, the wound continued

to close and the drainage-tubes could soon be dispensed with. But the patient suffered from disease elsewhere. Unfortunately, it had not previously been observed that the patient's urine was alkaline and contained a considerable quantity of pus. Catheterization showed a considerable distention of the bladder and retention of urine. Despite all kinds of irrigation, the bladder trouble became worse. Patient died six weeks after the operation.

At the autopsy, the external wound was found about healed. The dura and pia were adherent to the still recent connective-tissue cicatrix in the bony defect. In the right lateral ventricle a small quantity of reddish fluid. The pia covering the frontal convolutions is slightly opaque and gray; the convolutions are closely approximated. Upon cutting the frontal lobe of the right hemispheres into layers we see in its white substance a space filled with a reddish, soft structure, almost extending to the roof of the lateral ventricle. The bladder and kidneys show severe purulent cystitis and pyelitis. Upon pressure upon the calyces of the kidneys also purulent fluid escapes. I considered this kidney and bladder trouble as the cause of death.

The same scarcity of area symptoms in cases of abscesses in the frontal lobes also distinguishes those situated in the occipital and temporal lobes.

Among the large number of cases of abscess of the occipital lobe which have been observed, the characteristic area symptom for this portion of the brain—hemiopia—has only been described in one case, that of Janeway:[37]

A man of twenty-five had received a blow upon the left side of the head over the posterior parietal region with the butt of a revolver; this occurred November 4th, 1885, and resulted in a wound which suppurated and healed slowly. Six weeks later there was weakness of the left extremities, which increased to paresis, so that patient was confined to bed. There was constant headache since the injury. Upon admission to the hospital, there was a scar over the upper portion of the left parietal bone, anæsthesia on the left side of the face, arm, and forearm, also hemiparesis of this side excepting of the face and tongue. Patient was somnolent, excited from time to time, and complained of headache. Upon questioning him in a loud tone of voice, he answered correctly and without any

disorder of speech. There was complete and markedly developed left-sided hemianopsia. Ophthalmoscopic examination showed choked disc on both sides. Soon there was also a repetition of the evening rises in temperature, which had been absent since the time of the injury; and during the last days also slowing of the pulse. Three months after the injury Bryant trephined the right side of the cranium at a point corresponding to the cicatrix of the left side. Puncture with the needle of an aspirator did not reveal any pus. Then the left side was trephined in the region of the scar; here also no pus was found. Death nine hours after the operation. The autopsy revealed an abscess in the right occipital lobe, round in form, about two inches in diameter, situated in the white substance of the brain, near the posterior portion of the surface of the lobe, but yet separated from it by a layer of normal gray substance. The abscess had a distinct capsule and contained yellowish-green pus. The opposite hemisphere and the base were normal.

In the tuberculous abscess, not considered here, which Wernicke and Hahn (see above) diagnosed and opened, hemianopsia served as a very valuable point in the diagnosis of the localization of the area.

Modern brain-physiology places the centre of hearing for the opposite ear in the occipital lobes. If only the one ear be the seat of otitis media which has caused suppuration in the brain, and the other ear remains perfectly healthy and active, deafness of the latter would be a significant area symptom, at a time when other symptoms lead us to suspect the development of an abscess as a result of the ear trouble of the opposite side. Naturally in such a case the integrity of the peripheral apparatus of hearing must have been found preserved, by examination, as far as this would be possible. I do not know whether this symptom has ever been looked for in a suspected case. Since, according to Barr, in two-thirds of the cases chronic purulent inflammation of the middle ear is bilateral, it is evident how the presumable disturbance of hearing on one side may be overlooked.

The diagnosis of an abscess in the temporal lobe is, therefore, chiefly dependent upon the fever movement and the pressure symptoms. The uncertainty of the former is obvious and has been mentioned above. When suppuration in the ear

already exists, the disturbances due to the suppuration itself must remain unrecognized or overlooked. It will be understood, therefore, why the large number of the cases in question are not discovered before the autopsy and why others are only diagnosticated just before death by the symptoms of the last stage.

This was the case in one of those operated upon recently at my clinic and kindly sent to me by the well-known and experienced aurist, Dr. Hartmann. In giving the history I will follow the notes which he kindly left with me. H. H., thirteen years old, tall for her age and slender, anæmic, had had measles and scarlatina during her second and third years, without, however, any succeeding deafness or otorrhœa. About one year ago—June, 1886, she is said to have fallen into a cellar, falling upon the right ear, without, however, any hemorrhage from the auditory canal or any other effect visible in the ear. Since this time she says that she has suffered from a discharge from the ear and from headaches. The discharge from the ear was fetid and so profuse as to require frequent drying of the ear. Until July of this year she scarcely had any pain in the ear; but now these appeared and were so violent that fomentations and syringing out the ear with warm chamomile tea were resorted to. There being no improvement, she applied at the Hartmann Clinic, July 8th. Here the diagnosis of perforative middle ear inflammation was made; there was also marked swelling of the external auditory canal. A moderate amount of fever. 20th, chill, high fever, no vomiting, severe pain in mastoid process. 21st, swelling over the mastoid process. Ice applications cause some moderation. 22d, opening of the mastoid process. As soon as the integument was cut through, fetid pus escaped, especially from the upper angle of the wound. The bone itself was very rich in blood (osteitis granulosa), but there was no sequestrum and no great quantity of pus. The opening extended into the mastoid antrum, as proven by communication with the middle ear. On the twenty-third day after the operation there was a single attack of vomiting, which was ascribed to an error in diet. After this patient felt well, appetite good, no fever. The headaches, which had lasted an entire year, disappeared.

This favorable condition lasted about twelve days, after which the headaches returned and a feverish chilliness set in,

appetite became poor, no fever. Notwithstanding the attempt to keep the wound in the mastoid open by a leaden tube, it closed in the very shortest period. After August 13th there was, besides the chilliness, also repeated vomiting, and the patient became stupid. On this account the firmly-closed bone of the mastoid process was reopened extensively, but no pus was found. This time the general condition of the patient continued poor after the operation. The headaches constantly increased in severity. Constipation. Pulse rather rapid. No chills, no vomiting. Consciousness constantly preserved. August 20th, convulsive spasms in left leg. During the night the patient cries considerably. During the nights of August 21st and 22d she is suspiciously quiet, does not complain, lies as though asleep, answers still to questions in a loud tone in the morning. Pulse irregular; after a series of rapid pulsations there are others at suspiciously long intervals.

The patient was brought to my clinic at four o'clock in the afternoon, in order to allow a search to be made for the suspected suppuration in the brain. She is entirely unconscious but still moves her hands and feet. Shortly after five o'clock she sank into a deep coma and immediately ceased to breathe; pulse scarcely perceptible, but heart-sounds can still be heard plainly. Artificial respiration was resorted to; this caused the pulse to improve and to become full and regular.

While in this condition a piece of bone, measuring 5 cm. square, was chiselled out of a spot above and behind the auditory canal, about corresponding to the posterior inferior angle of the parietal bone. The unchanged dura bulges into the opening made by the trephine, but presents no pulsation. Incision through the membranes into the brain. Immediate escape of a large quantity of fetid, greenish pus. The abscess cavity was drained and an antiseptic dressing applied. About fifteen minutes after the operation, artificial respiration could be dispensed with, for the patient began to breathe again. During the first hour the respiration followed exactly the type of Cheyne-Stokes' phenomena. Pulse 60 to 64, full and strong.

At one o'clock in the night, breathing became more frequent and more shallow. Rattling was heard over the chest. At two o'clock patient died.

The autopsy demonstrated the existence of an abscess in

the right temporal lobe; it took in almost the entire lobe and had opened into the descending horn of the lateral ventricle.

No communication with the ear was found, nor was there any suppuration visible on the surface of the petrous portion of the temporal bone. There was no thrombus in the transverse sinus.

This abscess, which was discovered late because it had developed almost without exciting any symptoms, had been operated upon too late, for undoubtedly the rupture into the descending horn of the lateral ventricle had already taken place on the morning or afternoon of the day of operation. The deep coma and the cessation of respiration at the time of admission to the clinic prove this. Nevertheless, it is interesting to note that even after the rupture, evacuation of the abscess succeeded in starting up the respiration, which had already ceased.

Under similar circumstances I operated in another case, with the same unfortunate result; but I have not attempted the operation a third time. Both cases showed how rapidly the change from the latent to the terminating stage of an occipital abscess may occur. We are taught, therefore, to make up our mind rapidly in these cases; temporizing and further observation must be very limited at this time.

Of special interest in connection with the preceding case is one that was presented and discussed at my clinic July 17th, 1888. A man of thirty-two had had suppuration in the right ear since his childhood. For the last three weeks he complained of severe pains in the head and the ear; these had increased so as to become unbearable during the last two or three days. They were now located in the head, confined to the side of the affected ear. When brought to the clinic on the morning of the 17th of July, he seemed stupid and drowsy. The obscuration of consciousness progressed rapidly during the following hours. The pulse, which at first was 60, two hours after sank to 50, to 40 beats per minute. Patient shrinks upon percussion of the cranium between the right frontal and occipital regions. Patient looks very much weakened, seriously ill, and slightly jaundiced. Temperature 102°. Right pupil fails to react to light. Left facial seems paralyzed. Mastoid region not changed, no swelling, no œdema. Auditory canal not narrowed; it is full of pus. Drum-membrane

very red. During the transfer of the patient to the surgical clinic, at noon, he was in a condition of deep coma, tossing about from side to side, grinding his teeth together and groaning loudly. Suspended respiration as in the Cheyne-Stokes type. Right pupil dilated. Pulse small, 70 to 80 per minute. I explained in the clinic why I diagnosticated that a rapidly growing abscess in the right occipital lobe had already ruptured. The frequency of the pulse increased during the clinical examination, but it became smaller constantly. There were constantly longer intervals in the respiration, and in two hours the patient died. The autopsy confirmed the diagnosis. An extensive abscess, involving rather the posterior portion of the temporal lobe, had perforated into the lateral ventricle.

Macewen[38] also, in the first abscess which he found and opened, had to operate too late—*i.e.*, after perforation into the lateral ventricle had resulted. In the paper of this excellent surgeon read beore the British Medical Association at Glasgow, it is not stated that the case in question was associated with suppuration in the middle ear; but in the otological section of this same meeting, Barr (*ibidem*, p. 473) refers to 7 cases of abscesses of the brain in cases of otitis media, 5 of which recovered and 2 died. It looks as though one of these fatal cases were the one which is briefly sketched in the following lines. Notwithstanding the lack of intelligence on the part of the patient, paralysis of the oculo-motorius and of the muscles of the face and arm on the right side was made out. The operation was performed upon the patient while he was in an almost moribund condition. The membranes of the brain were very much congested, and three-quarters of an inch beneath the surface of the brain the abscess was found. After withdrawing several ounces of pus, a spherical body was seen floating upon the pus still remaining in the abscess cavity; this turned out to be an old encapsulated abscess around which the new abscess had probably formed. The patient seemed to have improved some after the operation, but died from exhaustion. The autopsy showed, besides the abscess cavity, also acute encephalitis of the second and third frontal and of the central convolutions.

I operated successfully and effected a permanent cure in a case which I presented at the Berlin Medical Society in December, 1888. It represented a typical example of suppuration in the temporal lobe and its diagnosis.

A turner, named Hoffman, of Berlin, twenty-nine years old, tall, thin and anæmic, had suffered from purulent discharge from the right ear since his fifteenth year. He was unable to state the cause. The trouble had not annoyed him much. The discharge had been profuse at times, scanty at others; sometimes it was quite purulent, at other times less so. He had occasional attacks of earache; these had been especially marked during the last three weeks; during this time he consulted an aurist, Dr. Hartmann, of Berlin; the latter sent him to the clinic. Besides having had an increased amount of pain in the ear during the past three weeks, he states that he has had attacks of vertigo, so that he staggered and ran against people in walking. In the evenings he felt quite ill and languid. He had lost his appetite and complained of feeling chilly and feverish successively. Since several days very severe headaches, continuing day and night and depriving him of sleep. He complains of pain over the entire head, more severe on the right than on the left side. At the examination upon admission to the hospital, he complained of feeling chilly and of the headache just spoken of. He gave the impression of being very weak and apathetic, and asked to be put to bed; in this position he could scarcely raise his head. When questioned he answered slowly, the answers being short and given with evident effort. His skin appeared slightly jaundiced and his tongue thickly coated. Temperature 103.4°. Respirations 24. Pulse 50 per minute. There were no noticeable disturbances of motion in the extremities. When asked to stretch out his arms, the left one would drop after a few moments, whereas the right one remained quietly in position. This was repeated every time that the attempt was made. Sensation, when pricked with a pin, seemed considerably diminished in the left arm and leg, as compared with the right limbs, and the pressure of his hand on this side was weaker, in fact scarcely perceptible. While being examined, twitchings began along the course of the facial nerve on the side corresponding to the affected ear. After this had passed away, the inferior branches of the nerve remained paralyzed. The tongue when put forward showed no deviation to one or the other side. The pupils were equally dilated and reacted to light. There existed no disturbance in the movements of the muscles of the eyes.

In the right external meatus was thick, ill-smelling pus. After this had been washed away, the depth of the ear was found filled with granulations, and the entire upper wall of the bony portion of the auditory canal was swollen and deeper than normal. The granulations bleed when touched with a probe. They can be raised up, and then the pus which lay behind escapes. Bare bone cannot be felt with the probe. The mastoid process is not swollen nor sensitive to pressure; but the granulations which fill the tympanic cavity are very sensitive. Pressure applied to the auricle does not cause much pain, but it is more painful if drawn backward and upward, as was done in introducing the speculum. Hearing is normal in the left ear, but in the affected ear—the right one—the ticking of a watch is not heard at all and moderately loud speaking only at a distance of three feet.

Upon tapping the head with the finger or with a percussion hammer, the pain is only increased when we encounter the lateral surface, namely, the squamous portion of the temporal bone, above the auricle. The patient complains of increased pain over a circumscribed spot in this region whenever it is percussed, but not when this is done over the mastoid process.

It was evident that the patient suffered from an old purulent otitis media which had lasted over ten years, and that it was now very severe. The deeper portions of the upper wall of the auditory canal seemed affected, judging by the swelling and by the situation of the granulations, which hung from above into the cavum tympani, and judging also from the fact that when the granulations were displaced a considerable quantity of pus, which lay behind, came into view in the upper part of the tympanic cavity. An extension of the suppuration to the neighboring cells of the mastoid process could not be proven; on the contrary, this could probably be excluded, on account of the absence of pain, swelling, and œdema of the skin over this part.

Was the suppuration of the ear sufficient to account for the fever? The patient stated that the exacerbation of fever began three weeks ago, and that since he had received regular medical treatment the quantity of pus has become less rather than greater. An extension of the process into the neighboring cavities did not exist. On that account, suppu-

ration had to be sought for in some other situation. The symptoms of fever and the accompanying disturbances were such as are found when pus collects and forms deep-seated abscesses: chills, increased malaise in the evening, a temperature of 103.5°, a dry, heavily-coated tongue, great thirst, great weakness and languor, with the feeling of being seriously ill. Since these symptoms accompanied a disease in the course of which and as a result of which the formation of collections of pus can occur, the symptoms of the fever indicated the existence of some such condition. Of course, a diagnosis of cerebral abscess could not be made from these symptoms alone. The pus might have spread into the interior of the ear, perhaps in the medullary spaces of the bone, or between the dura and the bone, or there might exist a beginning lepto-meningitis.

If, among other things, the symptoms of the fever also indicated the possibility of the existence of a cerebral abscess, the question would arise, What are the specific cerebral symptoms? A small abscess in a hemisphere of the cerebrum produces symptoms only when situated in the immediate vicinity of the motor area of the cortex. It is well known to what size an abscess can increase, as for instance in the frontal lobe, before it gives rise to a single symptom pointing to its presence. In the temporal lobe also we can only count upon signs of an existing abscess, when the volume is so great that the increase of size produces disturbances in the cavity of the skull due to the increased space occupied, causing a slowing of the circulation in the cranial cavity—which symptoms are usually called pressure symptoms.

The history of the patient on October 14th included severe headaches, which had begun very suddenly four days ago and were continually growing more severe and more troublesome; vertigo, staggering, and swaying were also symptoms of increased cerebral pressure, as was also the languor of the patient and his torpidity, his hesitating and evident difficulty in answering, including his desire to lie down and his inability to raise his head. But all these symptoms were so vague and so general, that they alone would not have indicated a condition of so-called cerebral pressure were it not for the slowing of the pulse, and especially such a slowing to only 50 per minute. It was observed in the clinic that in the course of a few hours the frequency of the pulse had fallen

from 64 to 50 and then to 46. In conjunction with the vertigo, the marked prostration, the drowsiness and headache, this symptom is certainly conclusive, for such a combination of symptoms, at least in this case, can only point to the existence of a disturbance which diminishes the space in the skull. Such a disturbance could only be a cerebral abscess, and a comparatively large one, in connection with and following the existing otitis media suppurativa chronica.

What was its situation? Large collections of pus cannot occur between the bone and dura, as shown by what has previously been said. Such collections are usually found with a suppurative process of the white medullary portion of the hemispheres. The question, therefore, was now limited to whether the temporal lobe or the cerebellum were affected.

The points which indicated the seat of the abscess to be in the temporal lobe were, first, the severe pain which caused the patient to shrink when percussed over the right lateral surface of the skull, second, the seat of the suppuration; confined to the cavum tympani, especially to its upper portion, and the integrity and absence of disease in the mastoid process.

It would certainly have been desirable to have had positive proof of the situation of the abscess; but the temporal lobe can be destroyed to a very large extent, as we have seen, without any function of the brain disappearing or being affected in any way. There are no positive cerebral symptoms which can be referred to the temporal lobe.

The fact that the left ear was healthy and that hearing was normal on that side did not justify us in assuming that the opposite temporal lobe was healthy; for the centre for hearing is supposed to be not in the white substance, but in the gray cortical portion; and we know that abscesses are apt to destroy the white matter and not the cortex.

In general, we may assume that a large abscess in the temporal lobe would increase the pressure in the entire cavity of the skull, by increased pressure of the cerebro-spinal fluid wherever this fluid is present, both in the arachnoid space and in the lymph spaces of the brain. Besides being distributed in an equable way by the cerebro-spinal fluid, this pressure is also propagated unequally by the soft but firm mass of the brain, so that those regions which are in the neighborhood of

the temporal lobe are influenced to a greater degree than those placed at a greater distance. As a result of this, we might imagine that whenever a large abscess exists anywhere in the right hemisphere, the functions of this hemisphere would be interfered with as compared with those of the unaffected left side. As a matter of fact, in our case the left extremities were affected, both as regards motion and sensation. The prick of a pin was felt to a less degree and was less painful upon the left than upon the right side; and when the left hand was raised it soon fell, whereas the right hand could be held out for a long time. These conditions were also considered by us as indications of affection of the right hemisphere.

We were not aided in the diagnosis of abscess by the existence of right-sided facial paralysis: for this nerve runs through the centre of the suppurating surface in the ear, and nothing is more probable and well known in cases of suppurative otitis than the introduction of irritation in the form of twitchings in the parts supplied by this nerve and of paralysis and paresis.

Accordingly, I diagnosed the existence of an abscess in the white substance of the right temporal lobe. If such an abscess had caused the symptoms of pressure already mentioned, especially slowing of the pulse, it must have reached a considerable size. If I allowed it further to increase in size, I would risk again being too late in operating. Consequently I proceeded to trephine at once.

For reasons which I shall explain below, I deem it proper to chisel open the skull rather more posteriorly than anteriorly. The upper border of the temporal convolution is indicated by the horizontal portion of the fissure of Sylvius, and in no case should the upper border of the trephined opening extend beyond this.

It is generally assumed that the temporal suture about corresponds to the course of the fissure of Sylvius. It is necessary, therefore, to mark out this suture in order to fix the upper boundary of the field of operation. For this purpose we draw a line about 5 cm. above and parallel to the zygomatic arch, which can easily be felt upon the living. Having marked the upper border, it is equally easy to find the posterior one. We construct a perpendicular to the basal line (that is, a line which connects the inferior orbital border with the

occipital tuberosity), starting from the posterior limit of the mastoid process and extending to the sagital suture; this represents the posterior boundary sought after. The anterior boundary corresponds to a line running parallel to the posterior through the temporo-maxillary articulation. Finally, we must take care that the lower border of the trephined opening is at least 1 cm. distant from the root of the zygomatic arch, which runs above the external auditory meatus. Thus we determine the limits of a space within which we chisel or bore, either more anteriorly or more posteriorly, according to circumstances. The opening should not be too small; it should be at least 3 cm. square.

In our case I made the trephined opening as far back as possible, extending a little beyond the posterior boundary line, and made it even larger than 3 cm. square. The exposed dura pulsated visibly and did not seem to be affected. It was incised, and immediately the extremely soft and evidently œdematous substance of the brain protruded. An incision into this caused a moderate amount of hemorrhage, but no pus escaped. It was difficult to separate the cerebral wound, which ran diagonally to the trephined opening and was about $2\frac{1}{2}$ cm. long, because the brain was so much softened and so fluid. The knife was inserted three times before the abscess was found lying in front of the incisions. At once about 30 ccm. of greenish-yellow, foul-smelling pus escaped. Digital examination revealed a smooth-walled cavity surrounded on all sides by soft brain substance. Into this cavity iodoform ether was poured, producing a minimum amount of pressure, and a drainage-tube was inserted to the depth of 4 cm. Around the drainage-tube iodoform gauze was packed loosely into the wound of the brain, and the wounds of the bone and soft parts over it were also packed with the same material. As a matter of course, the operation was performed under the usual precautions taken in this clinic. The entire head was shaved, then washed with soap and warm water, then with ether and alcohol, and then with sublimate solution; during the entire operation hemorrhage was completely controlled from all the bleeding vessels both in the tissues covering the bone and in the vessels in the pia. Finally the wound was covered with an iodoform tampon as employed by us so frequently in bleeding cerebral injuries.

Before the operation the ear was washed out as thoroughly as possible with a boric acid solution and the canal filled with powdered iodoform and iodoform gauze.

After the pus had been evacuated, the pulse rose from 50 to 54 and at the end of four hours to 84 beats to the minute. When the patient emerged from the chloroform narcosis, he stated that his headache had entirely disappeared; and it did not return. Late on the night of the operation, his temperature was 101.8°. On the 15th of November, after having passed a quiet night and having slept well, his temperature was 99.5° and pulse 68. The facial paresis still existed and the left arm tired very quickly. The dressings were changed daily; after the second day the iodoform tampon was left off and the size of the wound diminished by stitches placed in the angles, giving the wound as favorable a shape as possible. The drainage-tube was shortened after the ninth day and was gradually changed to one of smaller calibre. No return of the fever or headache; nor did any prolapse of cerebral tissue occur. The exuberant granulations were touched with nitrate of silver and were covered with small, short pieces of gauze kept in position by firm pressure. In less than six weeks the wound had cicatrized; the cicatrix had sunk deeply, as was shown, when the man was presented before the Medical Society in a perfectly healthy and robust condition. Up to date the patient enjoys perfect health; he resumed his work some time ago, although I undertook a second operation in December so as to effect a permanent cure for his suppurative otitis. This will be described at the end of this chapter.

Below I cite the seven cases which have been collected thus far of successful discovery and evacuation of cerebral abscesses; my case, just cited, was the eighth in this series. The histories are intended to present additional diagnostic points, as given by a combination of the different disturbances in the course of the disease, and especially by careful examination of the symptoms of cerebral pressure.

Schede is entitled to the credit of having been the first to perform a definitely-planned operation in an abscess following chronic suppurative otitis and of having effected an apparently permanent cure.

An architect,[39] twenty-eight years old, suffered for over half a year with suppuration from his left ear and attacks of ver-

tigo. He was admitted to the Hamburg General Hospital on January 12th, 1885. A short time before admission he had had a chill. The external auditory canal, which was very much narrowed by swelling and from which a reddish, shining vesicle protruded, was incised and syringed out regularly. Quinine was given internally. Patient had an icteric hue and complained of pain on the top of his head. The dressings contained very fetid-smelling pus. As the temperature was rising on the 14th of January, the mastoid process was chiselled open. From the deep portions pus exuded, and when the external auditory canal was syringed out there came away large pieces of dried (cholesteatomatous) pus; it was drained and packed with iodoform gauze; the dressings were changed daily. On the 16th of January the fever abated. His subjective condition was good. The wound showed no reaction. On the 24th, an evening temperature of 101.4°; had pain between the parietal and temporal regions. On the 27th temperature ranged between 102.2 and 103.4°; the granulations were scraped away until communication between the wound and the external auditory canal was re-established. On the 29th a paresis of the lower facial branches developed; there were peculiar disturbances in speech: patient makes use of only a few words, generally only "Yes" and "No," mixes correct words with fictitious ones, and reads in a stuttering manner; after having been told a word once he constantly repeats it in speaking; his memory is faulty; does not know how many children he has, what his name is, how old he is, etc. On the 30th there existed a still greater degree of aphasia. A rapidly-forming œdema developed behind the ear. He was trephined above and a little in front of the auricle. While operating, we noticed a small plug of pus, about the size of the head of a pin, lying against the bone about 3 cm. behind and above the opening of the external auditory canal; this pus escaped from a small fistula in the bone. The bone wall, which was extremely thin in this situation, was chiselled away, a piece the size of a twenty-five-cent piece being removed. The exposed dura was covered with red button-shaped granulations. Between these and the bone there lay flakes of vile-smelling pus. A still greater amount of bone was chiselled away in a downward and backward direction. When an exploratory puncture was made into the brain pus came away, and as soon as the dura

was opened, the pus poured out in such a quantity as to fill a cup; the size of the abscess was about that of a small orange. It was washed out with a 1:1,000 sublimate solution; drainage and dressings of sublimate gauze; on the following day, temperature normal. At first the patient slept a great deal, but improved rapidly. The return of the mental functions required more time; when questioned he answered incorrectly, and it was impossible for him to write; when he read he pronounced the first word distinctly after much hesitation, but after this he omitted the vowels and left out entire syllables. On the 12th of February drainage-tube was removed. On the 19th, 20th, and 21st of February he vomited, and pulse diminished in frequency to 60 beats per minute. Patient is perfectly stupid and has a pyæmic appearance. The dura bulged beyond the wound. Another incision was made and a quantity of thin, fluid pus, having a penetratingly fetid odor, was evacuated. Irrigation with sublimate solution. A drainage-tube 5 cm. long was inserted. The temperature remained normal. The mental functions were returning, though when reading or writing, but particularly when counting, he tired very quickly. On the 23d of March patient was made a dispensary case. Two months later, owing to fever, vomiting, and a slowing of the pulse, the wound had to be dilated and a new drainage-tube inserted. On the 4th of September, and again on the 16th of October, the patient presented himself fully cured and in a perfectly healthy condition. He talked fluently, but in the midst of a conversation referring to a certain subject—*e.g.*, to a tuning-fork—he hesitates and stops, for although he recognizes the tuning-fork as such, still he cannot say the word tuning-fork as rapidly as he would like; he called the attention of his physician to this condition.

Barker[40] reports:

A young man of nineteen, who had had scarlet fever when eleven years of age, suffered since then with a purulent discharge from his right ear. Toward the end of August, 1886, he felt ill and complained of very severe pain behind and around the affected ear. On the 11th of September he was compelled to go to bed; his temperature rose to over 102.2°, but sank on the following day. When admitted to the hospital all his mental functions were normal, as were also the movements of the eyes; both pupils were of the same size; there were slight

evidences of choked disc on both sides. A disagreeable-smelling fluid escaped from the right ear. Pulse 52. Morning temperature somewhat higher than that of the evening; after having risen once above the normal, it remained normal until September 21st. Examination of the ear revealed a large perforation of the tympanic membrane, with pus and detritus in the middle ear. The ear was syringed out with a quinine solution and filled with iodoform. There was neither sensitiveness nor swelling over the mastoid process. On the 21st and 22d the temperature was subnormal. Pulse 60. Increased amount of congestion and swelling at the point of entrance of the optic nerve. On the 25th the patient became stupid and gave confused answers. He suffered from nausea and vomited once. The right pupil is smaller than the left. Morning temperature elevated. On the 27th, the symptoms became more marked, especially the evidences of choked disc. On the 28th the mastoid process was trephined in order to discover whether pus existed in its cells; none was found, however. When the external auditory meatus was syringed out, fetid-smelling masses came out of the opening in the mastoid process. Drainage and iodoform dressings. On the following day there was slight improvement; temperature about 100.5°; the inequality of the pupils remained unchanged; the choked disc increased; patient felt better; had no pain. On the 2d of October, after a restless night, the patient again felt ill; had nausea and vomiting. On the following night had delirium and vomited; no headache; temperature normal. On the morning of the 3d he had a chill and temperature rose to 103°. He was then trephined, in order to find a suspected abscess in the temporal lobe. A V-shaped flap was formed, with its base above. The trephined opening was made three-quarters of an inch behind and the same distance above the external auditory meatus. The dura which lay beneath appeared to be healthy. When this was cut through, there came into view the apparently healthy-looking surface of the brain and a few drops of clear cerebro-spinal fluid. Barker inserted an aspirating needle in an inward, forward, and downward direction; from the depth of about half an inch foul-smelling, creamy pus was withdrawn. A forceps was introduced into the puncture, and by opening its blades the opening was dilated; from two to three drachms of pus were evacuated.

Drainage of the cerebral wound and of the cavity of the abscess was secured by inserting a tube to the depth of two inches. Iodoform dressing. On the days following the operation the stupor diminished and the optic neuritis remained unchanged; temperature still somewhat elevated. The rubber drainage-tube was changed to a silver one on the 7th; there was neither headache nor vomiting; mental faculties perfect; subnormal temperature; pulse 100. On the 12th the drainage-tube was removed; but owing to the fact that on the 13th an elevation of temperature and malaise occurred, a small drainage-tube was again inserted. The patient convalesced rapidly; no more pain. On the 19th the drainage-tube was removed. By the end of the month the choked disc disappeared entirely. In November, thirty-eight days after the operation, the patient left the hospital cured; there existed only a fistula in the mastoid process, which was purposely kept open with a fine silver tube. When he was discharged, it was determined positively that the hearing upon the left side was excellent. On the 4th of December the man again presented himself, fully cured and healthy in every respect.

Greenfield's case[41] was a difficult one as regards diagnosis, because at the time there was no discharge from the ear. However, it was ascertained that ten weeks previously the patient had complained of complete deafness, lasting five days; and on examination an old perforation was found in the left tympanic membrane.

The patient, a robust man twenty-six years old, had always been well, with the exception of scarlet fever during childhood. Ten weeks before admission, besides the deafness already referred to, he suffered from a cold and a cough lasting three weeks. After this he was well and able to work for one month. Two weeks previously he began to have headaches, lassitude and stupor, nausea and constipation. With these he has of late lost a great deal of flesh.

When examined after admission to the hospital, he lay with eyes closed and mouth open. When spoken to he answered with difficulty and in a stuttering manner, but otherwise correctly. There did not seem to be any disturbance of the intellect. He complained of headache, which he said had formerly been more severe. Pupils equal and contracted.

Tongue dry and fissured. Pulse 68. Temperature low. On the following day the left pupil became more dilated than the right. Patient appears to be getting more stupid; lies upon his face and bores his head into the pillow; later he lay upon the right side with knees drawn up, refusing to be moved.

Ophthalmological examination shows the left optic disc to be redder and more blurred than the right; in the course of time it became considerably more swollen and congested. Lips, teeth, and tongue are thickly coated. Pulse same as before. No elevation of temperature. On the 3d of January slight ptosis of the upper lid; very restless at night; increased malaise by day. Incontinence of urine. Pulse 112. No longer answers questions. On the 5th of January the patient refused to take food; the ptosis and dilatation of pupil are distinct on the left side. Optic neuritis on this side well marked, whereas the fundus of the right eye is perfectly healthy. For the first time a dirty brown-colored fluid was noticed escaping from the left ear. On the 6th of January general condition appeared to be better; the patient took food again. On the 8th of January the pupil was dilated ad maximum and immovable. Trephining at 2 P.M. Since the operator suspected the existence of an abscess in the anterior portion of the temporal lobe, he chose the temporal region for his field of operation. After the fibres of the temporal muscle had been divided, the trephine was placed upon the external surface of the temporal bone, one inch above the zygomatic arch. The trephine button consisted of a portion of the squamous portion of the temporal bone and a small piece of the wing of the sphenoid. On its inner surface there existed a distinct groove for a branch of the middle meningeal artery. The exposed dura bulged forward, appeared to be very tense, and did not pulsate. After incision of the dura, the knife was sunk into the brain in a forward direction for half an inch; immediately fetid pus was evacuated. The opening was dilated with a pair of forceps and a drainage-tube inserted, from which about two ounces were discharged. Antiseptic dressing.

After the operation the patient remained in a sleepy condition and only gave inarticulate answers. Alternating with this condition there was great restlessness and constant talking. On the 9th of January he attempted to remove the

dressings, but he answered the questions which were put to him properly; headache; the ptosis had disappeared; during the day the patient slept a great deal and became intelligible. When boric acid was injected into the ear it was discharged through the trephined opening. On the 10th of January the ophthalmoscopic examination revealed a contraction of the pupil and a distinct diminution of the swelling at the point of entrance of the optic nerve; vision very good; there was rapid improvement; the discharge became less and lost its disagreeable smell; the optic neuritis disappeared entirely. As early as the 13th of January the fluid which was injected into the ear no longer appeared in the wound. The drainage-tube was removed on the 23d of January. Complete closure of the wound on the 25th of January. The patient remained in bed until the 30th; he had sat up in bed as early as the third day after operation. Temperature normal. No trace of mental disturbance; on the contrary, well-developed intellect. On the 7th of February he presented himself fully cured. There remained only a slight purulent discharge from the left ear. Examination of the eye showed the left papilla to be paler than the right and less defined in its contour on the nasal side.

Macewen[42] took charge of the case of a boy nine years old suffering from a purulent otitis media occurring in the practice of Barr. A month prior to the operation he complained of pain in his right ear, which made him fret and prevented him from sleeping at night. His skin was hot and dry. He vomited, and after this became somnolent. The pain in the ear, the fever, the drowsiness, and the occasional vomiting were the prominent symptoms during the first seven days of his illness. On the eighth day he had a rigor which lasted a quarter of an hour. This was repeated about six times at intervals of about two days. On the nineteenth day of the disease he was admitted to the hospital. He looked pale and emaciated; he had a short cough; temperature over 102.2°; pulse 108; he was very languid and drowsy and complained of pain over his right ear, from which escaped a moderate amount of offensive discharge; Barr found a perforation in the upper part of the tympanic membrane; there was neither redness nor swelling over the mastoid process to indicate retention of pus, which would account for the fever. Barr opened the mastoid process and also the posterior wall of the external

auditory canal by chiselling. In irrigating the wound he succeeded in washing away some caseous débris. A drainage-tube was inserted. On the twenty-ninth day of the disease, after repeated chills and periods of fever, a copious discharge from the ear was noticed. The patient's condition was miserable; in fact, his enfeebled condition had grown more evident than before; he lay with his eyelids half-open, as if asleep, taking no interest in anything, and would not answer questions; lips and tongue dry and fissured, the lips being covered with sordes. The veins over the right side of the head and face were congested, as was also the external jugular vein. There was no hard, cord-like feeling nor any tenderness along the course of the internal jugular. When digital pressure was applied over the region of the right temporal bone, he complained of very severe pain; this was not complained of over other portions of the head. Frequent cough, with purulent, foul-smelling expectoration. Moist râles over both sides of the chest. Obstinate constipation. Trephining on the thirtieth day of the illness. A half-inch disc of bone was removed from the squamous portion of the temporal bone at a point one inch above and half an inch behind the centre of the external auditory meatus. The bone was normal. The dura did not pulsate. After division of the latter, brain substance immediately bulged into the wound. A hollow needle was inserted in an inward and backward direction; after it had penetrated the brain substance for about three-quarters of an inch, there was a sudden escape of foul gas accompanied by a bubbling sound and a few drops of pus. The needle was inserted a little further and about twenty grammes of pus escaped. The opening was now enlarged by forceps, and portions of the brain-tissue which had become necrosed were removed. Macewen made a counter-opening into the base of the skull, entering the upper wall of the auditory canal. The dura here was also found intact; as soon as it was divided, pus poured out. From this opening the abscess cavity was washed out with a solution of boric acid, the solution running out through the first opening. Two drainage-tubes were inserted, one above, the other below; the parts were thickly dusted over with powdered boric acid and dressed with sublimated wood-wool pads. There was a marked improvement during the first week after the operation; fever ceased, intel-

lect became clearer, and the patient showed an interest in his surroundings. Once a week the dressings were changed and the drainage-tube shortened. At the end of five weeks the last drainage-tube was removed. The child rallied, appetite was good, and the boy improved rapidly. The purulent discharge from the middle ear had not ceased at the end of eight weeks. Hearing was diminished, but still comparatively good. Three months later the child died, death being due, as stated by Barr at the Glasgow meeting, to a tubercular peritonitis.

The autopsy revealed a complete cure of the abscess by means of firm, cicatricial tissue, which was, however, small in amount.

The case of cerebral abscess diagnosticated by Ferrier[43] and operated upon by Horsley[44] is of especial interest:

A man forty-seven years old had an affection of the left ear, lasting fifteen days. On the fifth day of the illness a purulent discharge was observed. Numbness and a pain on the left side of the head, increased by percussion, especially over the frontal portion. Photophobia. Pulse 52. Improvement during the succeeding days after syringing out the ear. During the third or fourth week of the illness about the same condition existed, excepting a greater desire to sleep. At the end of the third week there was difficulty in speaking and the use of wrong words; he named articles incorrectly and became irritated when not understood. The right angle of the mouth could be moved less energetically than the left. Strength of right arm less than that of left; with the left he could raise one hundred pounds, with the right less than eighty pounds. Tendon reflexes good on both side. No facial disturbances nor of movements. Optic neuritis on both sides; both pupils equal. Hearing good in right ear; but left only hears watch on contact. Examination of the ear was incomplete on account of tenderness and swelling. He had no headache, but when percussed there was pain over a point exactly two inches above the external auditory canal. There was no vomiting, no convulsions, no fever. The diagnosis was based upon the paresis of the right facial in conjunction with the disturbance of speech, which necessarily had to be referred to a diseased area in the anterior portion, anterior third, of the temporal lobe, near Broca's centre and the centre for the facial nerve. In addition to this there existed the tenderness over the bone corresponding exactly to this position of the abscess.

In the beginning of the fifth week of the disease, Horsley operated, making a T-shaped incision, the arms of which joined over the painful spot. The temporal muscle and periosteum were pushed away from the bone. The button of bone which was removed from the skull measured about one inch in diameter. The dura did not pulsate, but was so hyperæmic as to look almost purplish. It was incised, and immediately œdematous brain tissue bulged out. A trocar having a diameter of 3 mm. was introduced to the depth of 1 cm.; this resulted in the evacuation of more than half an ounce of pus. It had no odor and resembled milk. A silver drainage-tube was left in the wound without washing it out. Up to the point where the drainage-tube came out, the wound in the skin was closed with sutures.

A rapid cure followed without any complications. There was elevation of temperature on the second day after the operation only. On the fourteenth day the drainage-tube was removed. A few days after the operation the disturbance in speech began to lessen. In the third week the paresis of the right facial nerve had disappeared. At this time, also, no traces of optic neuritis could be found. The discharge from the ear had ceased. A cicatrix could be seen in the tympanic membrane.

Regarding etiology, it is worthy of mention that an acute otitis media was the cause of an abscess in this case; that the abscess developed very rapidly; that the pus was of good quality, without any signs of decomposition, and yet it must certainly have been highly infectious.

During the past year Barker[45] opened another abscess of the temporal lobe with excellent results. It occurred in an engineer thirty-three years old who had acquired a right otitis media following scarlet fever during his seventh year; since then he has had a discharge from the ear; this discharge would disappear for a time and then return again. At twenty-one he commenced to have epileptic convulsions and at twenty-four he was treated for an ulcer of the stomach. At twenty-nine he injured himself by a fall upon his back, the consequence of which was motor and partly sensory paralysis of the right arm and leg, lasting eighteen months and followed by recovery. Since May, 1886, he has complained of weakness and a chilly feeling in the right leg, which he dragged after him in

walking. On the dorsum of the right foot there existed a localized anæsthesia. On account of these symptoms, lateral sclerosis of the spinal cord was suspected. Under proper treatment all these disturbances disappeared. In December, 1886, there developed a right-sided facial neuralgia. Until December 5th, 1887, he did not seek medical aid, but then, on account of increased discharge from the ear, he placed himself under the care of an aurist. The right tympanic membrane presented a large perforation; insufflation of powdered iodoform. On the 28th of December there was pain over the entire right side of the head; no swelling over the mastoid process; no changes in the optic disc; normal reaction of the pupils; temperature somewhat elevated; neuralgia of the trigeminus, especially at night. January 20th, vomiting. When the right side of the head was touched, especially in the region behind the mastoid process, great tenderness was found to exist. Patient was somewhat stupid and only gave hesitating and incomplete answers regarding his condition. On the evening of the 23d violent headache and vomiting. At night two epileptic attacks in less than an hour, during which the right side was mostly affected. Temperature in the morning, 99.8°; evening, 101°. In order to discover whether there was retention of pus in the ear, the mastoid process was opened by chiselling, but nothing was found. On the 28th, greater enfeeblement and sleeplessness. On February 1st a general diminution of strength; the skin of an earthy color; a very sick appearance; urine voided unconsciously; slight diminution in strength in the left facial nerve. On the 2d of February the stupidity became more marked, the right pupil larger than the left. The arm became powerless, the extensors of the fingers and hand distinctly paretic, and with these also twitchings of the left hand. Pulse 64. Operation on the 3d of February. On account of the paralysis of the left facial nerve and arm, the crown of the trephine was first placed over the motor region, two inches below the sagittal suture and one and one-half inches above the guide to the fissure of Sylvius. A second button was removed a short distance below the first, and the bone lying between these was removed. At this point a puncture was made downward into the depth of the brain but did not strike pus. Then a third trephined opening was made, below a line corresponding to the

fissure of Sylvius, and posteriorly on a perpendicular line drawn from the posterior border of the mastoid process to the sagittal suture (one and one-half inches above the basal line and one and one-quarter inches behind the external auditory canal). A hollow needle inserted here allowed the escape of half an ounce of odorless pus. The puncture was enlarged by means of a pair of forceps and a rubber drainage-tube was inserted. When pressure with a sponge was made over the upper trephined opening more pus escaped. Hemorrhage from several of the veins of the pia was controlled by the Paquelin thermo-cautery and the wound was closed by drawing the skin flaps together and sewing them. An improvement was observed at the end of three hours; pulse 140. On the 5th of February the improvement as to the facial paralysis was well marked; the drainage-tube was changed; the rubber tube was replaced by a silver one. The weakness in the left arm became less. The headache, though less severe, still persisted. In a few days the paresis of the left hand also disappeared. By the 10th of February the headache had disappeared. On the sixteenth day after the operation the patient got up, feeling very strong, and on the 8th of March he was discharged from the hospital. He had occasional neuralgic pains. The last time he visited his physican, on the 28th of March, he still complained of headache, but looked very well and had an excellent appetite.

Macewen [16] also had a second case of abscess of the temporal lobe (following suppurative otitis media) upon which he operated successfully. This short account of the case is taken literally from his Glasgow address:

"The patient, who showed symptoms of a cerebral abscess, had left-sided ptosis, mydriasis, and paresis of all the muscles of the eye, with the exception of the external rectus. The facial muscles were paretic upon the right side, still the power of mimicry existed to a certain extent, and the right eye, which when asleep remained half-open, could be closed at will. Besides this, the right arm was distinctly paralyzed. The lower extremity showed no motor disturbances, and the sensation in the skin was not diminished in the paralyzed portions. In consideration of this complex chain of symptoms, the existence of an extensive lesion, involving both the course of the oculomotorius and that of the inferior portion of the central convo-

lution, could be inferred. It was, however, evident that there could be no extensive destruction of the central convolution, for were this the case, the centre for the lower extremities would also necessarily have been involved; this was not, however, the case. A lesion of the crus cerebri also had to be excluded, for any pressure in this region, direct or indirect, would have led to serious disturbances and the symptoms would have had to become developed in the reverse order—namely, the lower extremities would have been affected first and the motor disturbances in the face would have been the last to appear. Neither could the internal capsule have been the seat of so extensive a lesion, for then hemiplegia with affection of Charcot's bundles would have been produced. The paresis of all the muscles of the eye could only be attributed to a disturbance seated in the origin of these nerves; the occurrence of paresis of the face and arm also was such that we had to suppose an ascending pressure—a pressure increasing from above downward. The only situation which could be the source of all these disturbances would have to be in the temporo-sphenoidal lobe. Therefore an incision was made in this region. In the white substance of this lobe an abscess was actually found; three ounces of pus were evacuated and there was a resulting disappearance of the symptoms which have been described. At the end of three weeks the wound had healed completely." [47]

These histories of the cases illustrate thoroughly the difficulties in making the diagnosis. However, in eight cases a correct diagnosis was made and the life of the patient saved. Consequently there is every reason for investigating all causes leading to a recognition of the deep-seated degeneration in these successful cases.

In cases where the fever was the prominent symptom, the opening of the mastoid process preceded the operation upon the brain, because it was impossible to exclude the possibility of the retained pus in its cells and antrum being the sole cause of the rise of temperature. This was done four times—in the cases of Schede, Barker, and in the first one of Macewen's. Once only (in Schede's case) was pus found in this situation. When no pus was found here, the cause of the fever had to be sought for elsewhere in a retention of pus in some other situation, either a collection between the dura and

the bone, or else in the brain itself. It is evident that where only fever and headache in the vicinity of the ear exist in connection with an old aural discharge, we would think of a spreading of the process to the interior of the ear—to divisions and spaces which had not been attacked before; this conclusion cannot be avoided when at the same time there is an obstruction of the external auditory canal, as in Schede's case, or swelling and pain over the mastoid process, as in Barker's first case. In this connection and in all other parts of these pages I have excluded Macewen's second case, because I was only able to obtain a report of the existing, though important, nodular symptoms. In almost all the cases, we observe that the patient complains of being very ill, is very weak, dull, and languid, emaciates rapidly, loses his appetite, and has an earthy-colored or icteric appearance. The severe and excruciating headaches are constant symptoms; in only one case were they absent for a time, or less severe than in all the other cases (Horsley). Five times it was distinctly stated that when the affected side of the head was percussed over a distinctly localized spot, generally just above the upper border of the concha, a sharp pain was felt (Macewen, Horsley, Barker, v. Bergmann). Furthermore, in the majority of the cases (Barker, Greenfield, Macewen) there existed nausea and vomiting. In every case there was a somnolent condition, drowsiness, and stupidity, or even unconsciousness (Greenfield). A diminution in the frequency of the pulse received special mention in three cases (Horsley, Greenfield, and v. Bergmann). It is a very important fact that in some cases changes were found in the optic disc by an ophthalmoscopic examination. This was limited to the affected side in two cases, and consisted of a rapidly-forming choked disc (cases of Barker, Horsley, and Greenfield). In my case it was absent; in several of the cases this particular examination does not seem to have been made. In two cases the pupils were wider upon the affected than upon the healthy side (Greenfield and the two cases of Barker).

Rise of temperature in cases of old suppurative otitis media is a very common occurrence. When it occurs with evidently recent manifestations of exacerbating inflammation either in the external auditory canal or in the mastoid process, we are justified in considering these the causes of the elevation

of temperature, just as in those cases the increase of the discharge from the ear is accompanied by rigors, elevation of temperature, and headache. Cleaning the ear, causing a diminution of the swelling in the external auditory canal, and the removal of obstructing granulations are the means adopted to overcome the inflammatory affection and to bring the condition back to the normal quickly.

If in reference to the diagnosis of abscess from the fever symptoms we only obtain a small amount of information, then it becomes necessary for us to depend upon the increasing symptoms of cerebral pressure, which occur as a result of the tension made by the contents of the abscess. The differentiation between abscess and tumor is apt to depend solely upon the cause of the disease—that is, whether a suppurative otitis media exists (Barker's second case). Where the otitis has ceased or is only recognizable very late in the disease, as in Greenfield's case, the difficulty in making the diagnosis will be apparent. When, after thorough evacuation of and free exit for the pus from the ear and the cells of the mastoid process, fever, together with the symptoms of pressure, still continues, we have that combination of symptoms which leads us to make a diagnosis of abscess in the temporal lobe. In Barker's and Greenfield's observations, the increase of the choked disc, as time advanced, was certainly the most important symptom. To this must be added the variations in the intensity of the symptoms, which is particularly striking in the cases of Greenfield and Horsley. At one time the patient was entirely unconscious, answered no questions, and took no food; then again, on the following days, his mind was clear, he ate and drank. At times the frequency of the pulse was markedly diminished and at other times 112 or more.

The epileptic or the epileptiform attacks, which were so frequently spoken of in the description of other abscesses of the cerebrum and are regarded as initial symptoms, seem to be absent in cases of abscesses of the temporal lobe, or to occur only exceptionally. In the second case of Barker's, which was so complicated, the epilepsy was probably independent of the affection of the temporal lobe.

Area symptoms are specially mentioned in 5 of the 8 cases of opening of abscesses. Schede and Horsley's observations emphasize one symptom, which Wernicke in his description of

disease of the temporal lobe gives a great deal of importance to. The occurrence of complete aphasia was preceded in Schede's patient by a disturbance of speech, which consisted in the use of wrong words; and in Ferrier-Horsley's description it is stated that the patient misnamed articles. Where patients use improper words in place of those they wish to say, there exists a condition known as conductive aphasia, which assumes an interruption of the association tracts, an interruption which may be due to a destructive process in the temporal lobe; consequently, in these cases it is due to a suppurative destruction.

But there is still another symptom which, in a certain sense, must be looked upon as an area symptom. As a rule, we may assume that a large abscess in the temporal lobe increases the pressure throughout the entire cavity of the brain by increased tension upon the cerebro-spinal fluid, wherever this fluid lies in the arachnoid sinus and in the lymph-spaces of the brain. But besides the equable pressure of the cerebro-spinal fluid, the pressure is carried along still further by the firm, or more correctly firmly soft mass of the brain; but the latter is naturally unequal, so that the province of the brain in the vicinity of the temporal lobe is affected more than those portions lying at a distance.

It would seem, therefore, that whenever a large abscess exists in any part of the right hemisphere, the functions of this hemisphere would be diminished in comparison with the normal left hemisphere. This did occur, as a matter of fact, in my case, in Horsley's case, and in the second case of Barker's. In my patient the hand on the opposite side of the affected ear fell motionless whenever it was raised, whereas the right hand could be held in an extended position for a long time. Sensation seemed to be diminished on the left side, for the prick of a pin produced much less pain on the left hand and arm than it did on the right side. In Barker's second case and in Horsley's case not only the opposite arm, but also the facial nerve of the unaffected side was paretic.

These hemipareses were most marked in Macewen's second case. In this case there existed, in addition, the interesting paralysis of the oculo-motorius, which was referred to a disturbance affecting the origin of the nerve.

In all of the cases the lower extremities were not affected,

showing that the lower parts of the central convolution were the portions affected. This condition goes to prove that the pressure symptoms extended from below upward, extending unequally through the solid mass of the brain, those tissues lying near the abscess in the temporal lobe being more affected than those situated at a greater distance.

In cases of formation of pus between the dura and the bone, the general and circumscribed effects of pressure just mentioned have not been observed. On the other hand, the guides to diagnosis were the marked changes over the portions of the bone affected in these cases.

A consideration of these conditions might enable us to differentiate between deep cerebral abscesses and accumulations of pus between the dura and the bone before opening the skull. In the successful cases of the latter variety there generally existed a fistulous passage which led into the skull; by enlarging this with a chisel or sharp spoon, the spot where the pus was retained was reached.

Such was the case in the first report of the opening of an intracranial abscess by Schondorff.[48] The case occurred in a farm hand, twenty-four years old, who had had trouble with his ears previously until July, 1883. While serving as a soldier, after having been in swimming he became affected with a throbbing pain, ringing in the ears, and difficulty of hearing, without any discharge. In August the pains had gradually subsided; in September the buzzing and drawing sensations were increased upon coughing or changes in the weather. End of October, fever and boring pains; these symptoms ceasing after a profuse suppuration had suddenly appeared. On the 2d of February, 1884, he was admitted to the surgical clinic in Greifswald. Had left-sided headache and severe vertigo; no fever; marked and continual buzzing; there was difficulty in hearing and a profuse, thin otorrhœa; Eustachian tube patent. A small polyp was seen at the tympanic membrane and was removed; the tympanic membrane was then incised so as to facilitate the escape of pus. A large quantity of a four-per-cent solution of boric acid was injected through the catheter, although the water came away clear; still, after the washing was stopped, in a very short space of time the entire auditory canal, as far as the concha, was again filled with pus. The condition of the pneumatic cells of the temporal

bone did not explain the pus, nor did the appearance of the thickened mucous membrane of the tympanic cavity account for the blennorrhœa. On the 23d of February he was free from pain and was discharged from the hospital, not as a cured case, however. He returned on the 3d of March; fever (103° noon) with frequent attacks of vomiting, which had kept up during the two previous days; constipation, slowing of the pulse, pale and sunken appearance, but above all, frightful fixed headache over the lambdoid suture at the posterior inferior angle of the parietal bone. Objective examination of the ear same as before. The large quantity of pus was foul-smelling: after lying on the right ear for some time, there was a discharge through the nose and mouth. Mastoid process unchanged and not at all sensitive. When light pressure was made upon the spot on the left parietal bone already mentioned, where later on a portion of the scalp, having a circumference of about a five-cent piece, had a doughy feel, the patient fainted. Diagnosis: retention of pus in the mastoid process, with meningitis incipiens. After the middle ear was washed out, as described above, the pain ceased and the fever diminished, but not the suppuration. On the 10th of March, temperature 101° P.M.; violent headache, which could not be relieved by the application of ice and painting with collodion over the mastoid process. On the 12th of March the mastoid process was chiselled open, and then a broad speculum measuring 25 mm. in length and 15 mm. at its base was passed through the sclerosed bones in the direction of the antrum. A discharge of pus could not positively be made out. Next the posterior bony wall of the auditory canal was removed with a chisel and hammer, and the wound was then washed out through the auditory canal; but again no discharge of pus. The wound was dried with five-per-cent chloride of zinc cotton, a drainage-tube was inserted surrounded by iodoform gauze, boric acid was gently blown into the auditory canal, and Lister's occlusion dressing applied.

On the next and the following eleven days there was slight remittent fever, severe vomiting twice, vertigo, fainting, and violent headache on the left side posteriorly. When the dressings were changed on the 13th of March, a small quantity of pus was found in the auditory canal; the wound in the bone in the mastoid process was dry except a small drop of pus in

the middle of the posterior wall of the artificial funnel, and below this a small fistula was discovered. This was enlarged with a sharp spoon and a bullet-borer; then a sound was inserted obliquely for at least 2 cm. backward and inward; this was followed by the discharge of about 60 grammes of pulsating pus, which squirted out when the patient coughed; the pus was then washed away with a solution of carbolic acid. Although the intracranial abscess was now recognized as the cause of the pain when pressure was applied to the above-mentioned spot, as also of the œdema spoken of, still a further operation was not attempted until the 11th of April, because in the mean while, when the dressings were changed on the 14th of March, all the objective and subjective symptoms had disappeared; the fistulous passage, in spite of the daily employment of the sharp spoon, was constantly covered with granulations; gradually rigidity in the muscles of the neck developed, as did pain on pressure over the spinous processes; also difficulty of motion and numbness in the arms. To prevent a threatening meningitis, the fistulous passage was transformed with a chisel and mallet into an opening, extending through the entire thickness of the skull, the floor of which was formed by the thickened, pulsating dura. The occipital artery had to be cut through. Owing to the fact that the wounded bony canal crossed the transverse sinus at the posterior parietal angle, the dura, which pulsated strongly, was irrigated with a solution of carbolic acid and lightly dried with chloride of zinc cotton; the entire cavity was then filled with iodoform gauze; Lister's occlusion dressing was placed over the wound.

All threatening symptoms disappeared permanently, and the large skin and bone wounds were healed, after nine dressings, on the 5th of June; no further secretion from the tympanic cavity. When discharged, on the 24th of June, the patient had neither headache nor buzzing in the ears and heard very well; on the tympanic membrane a cicatrix could be seen.

A case which was similar to this one was lately published by Hoffmann,[49] the sixth and last of his cases of external purulent pachymeningitis. A feeble girl, one and one-half years old, was affected with a left-sided otorrhœa during an attack of scarlet fever. One month later a decided swelling formed

over the mastoid process of the affected side, which, when the patient was admitted to the hospital two months later, presented an abscess as large as a hen's egg extending down the neck. After the abscess had been opened, a fistula leading vertically into the interior of the bone and lying 1½ cm. behind and ¾ cm. above the upper wall of the external auditory canal was discovered. It was enlarged with chisel and sharp spoon. The operator then entered a cavity which apparently lay within the skull. A thick sound could be passed, without any force, upward and forward into the skull to the depth of 11½ cm.; in passing it upward and backward it could only be introduced to the depth of 2 cm. The bony passage was enlarged to the size of 2 cm. in diameter. At the bottom of the exposed surface the pulsating dura covered with pus was seen. A tablespoonful of pus escaped from the skull during the chiselling. It was not possible to discover any communication between the wound and the middle ear. A drainage-tube covered with iodoform gauze was inserted into the lumen of the opening, and the external abscess cavity was tamponed with the same material. The drainage-tube was removed on the fourteenth day, and eight days later the wound had healed.

The discharge from the external auditory canal disappeared at once. The child died two months later, owing to increase in the severity of the cough and general debility.

Ceci[50] was compelled to follow a lengthy and difficult route in order to permit of the escape of pus over the tentorium. This history, of which extracts are given, is therefore a very interesting one, for it describes disturbances which are peculiar to cerebellar abscesses, and may therefore be regarded as the only example of successful opening of the skull in a case of cerebellar abscess. In addition, the observation also furnishes an illustration of the route of suppuration from the middle ear to the occipital lobes.

This history is one of a laborer, thirty-nine years old, who claimed to have suffered only from an eruption which was regarded as syphilitic while a soldier, and also from anthrax and suppuration of lymphatic glands. Present trouble was said to have originated in a swelling of the left mastoid region three years previous; this swelling broke and much pus escaped. At the same time an annoying tinnitus began to affect

the ear of the same side, and after disappearance of the swelling the tinnitus continued. Some time afterward a few drops of pus escaped from the ear. Three months later there was a recurrence of the suppuration over the mastoid; incision and scraping of the mastoid. Hearing had been almost entirely lost. A fistulous passage which led to carious bone remained. Twenty months later, after the patient had recovered his hearing and the tinnitus had ceased, another abscess formed in the parieto-occipital region; it was incised and fetid pus was evacuated. Here also a fistula which secreted a small amount of pus remained. This condition of the patient lasted about a year. Upon consulting a physician, the latter used a probe, and passing through the fistulous opening for a distance of 1 cm. in a vertical direction, must have reached the cavity of the cranium.

Upon admission to the clinic, May 15th, patient was suffering from disturbances of locomotion causing rapid tiring, especially at dusk or in the dark; at such times he reels like a drunken man and must seek support from the walls in order to get along. At times he is annoyed by left-sided tinnitus and very intense headache in the temporal region. These symptoms increase when the pus seems retained in the fistulous passage, and diminish when there is a profuse purulent discharge. Once, after extraordinary exertion, the pain and disorders of locomotion became especially marked. At that time probing resulted in the evacuation of large quantities of pus from the fistulous canal and consequent improvement, lasting some time. He also complained of vertigo, especially when assuming the erect posture from a stooping one. He could not grasp small objects with the left hand with any degree of certainty. Formication of the left hand. For several days past, pains in the legs, radiating from the course of the sciatic nerve, more in the right than in the left; also sensitiveness in the elbow-joints. Feeling of heaviness in left parietal region. No paralyses in face or in ocular muscles. Left pupil somewhat larger than right. No other disturbances: no tender points along the spine. Absence of tendon reflexes at the knee and foot. The watch is heard at 50 cm. with the left ear, at 200 cm. with the right. Smell normal. Taste seems less acute on the left side of the tongue. Patient sways when requested to stand erect. He can stand upon one leg only a

short time and would fall if not supported. In walking he puts feet down in a decided way, but supports himself chiefly upon the right foot. He cannot walk along a straight line and deviates toward the left. In turning he sways and must spread his legs far apart so as not to fall. All these disturbances are increased in the dark. Sense of touch and of temperature are alike on the two sides and are normal. Since the fistula was placed pretty nearly at the posterior inferior angle of the parietal bone, it must have led either to the insertion of the tentorium or to its upper surface up to the third occipital convolution or the sulcus lying immediately above this. The diagnosis also considered the involvement of the cerebellum through pressure upon it by the collection of pus.

Operation after cocaine anæsthesia. T-shaped incision. Separation of periosteum. Chiselling open of the bony wall so as to form a funnel-shaped opening 20 mm. in diameter. The bone was sclerosed to a thickness of 25 mm. The probe introduced still further apparently lay on the tentorium above the transverse sinus. Next a large-sized crown of the trephine was applied above the opening. After removing the button of bone, evacuation of about 60 grammes of fetid pus. At the bottom of the opening cerebral convolutions covered with pus were found.

The case progressed without fever. Daily change of sublimate dressing. After several days the opening had improved considerably; healthy granulations had developed. Little suppuration. On the 15th of June, while changing the dressing, a considerable evacuation of pus from the bottom of the wound. Enlargement of the opening by means of a laminaria tent. June 20th, no swaying when standing straight in the erect posture. Certain gait; can turn without difficulty and can stand upon one leg. The disturbance in the sense of taste has disappeared. Certain grasp of objects. August 20th, re-examination confirming all these improvements. A small fistulous canal still remains at the seat of trephining.

The position of the pus immediately above the transverse sinus allows no other interpretation than that of a periphlebitis—an abscess about the walls of the vein. The pus, however, extends along the thrombosed vein far into the interior of the skull; in this case it extended to above the tentorium. A large portion of extra-dural abscesses in the cranium is

undoubtedly of periphlebitic origin. But this should be no reason for failure to evacuate them. On the contrary, we are in possession of a whole series of reliable observations showing that thrombosis of the transverse sinus is robbed of its dangerous character by removal of the pus.

Hoffmann (l. c.) cites such cases and increases the number by a very instructive one of his own observation (Case I.).

A boy of fourteen was attacked with an acute inflammation of the middle ear on the 8th of August, 1886. Upon admission to the surgical clinic of Greifswald, he had already become stupid and his fever was very high (105.8°); on this account the drum-membrane was incised; bloody pus escaped. Also immediate incision over the mastoid process. On the nineteenth day œdema and tenderness on pressure were present next to the last-mentioned incision. On this account the mastoid process was chiselled open. After removal of the outer lamellæ of bone, there was an escape of pus; this could be traced to the antrum, a funnel-shaped opening existing; fluid syringed into this escaped through the external auditory canal. A drainage-tube covered with iodoform gauze was inserted. On the second day after the operation patient had a chill, and fever rose to above 106°. On August 24th, during the change of dressing, a small opening was discovered in the posterior wall of the opening in the bone; pulsating pus escaped from this small opening. With a sharp spoon the fistula, which ran in a backward direction, was enlarged; several grammes of pus were evacuated in this manner. During the following days there were repeated chills. A painful swelling is seen on the left side of the neck at the anterior border of the sterno-mastoid; this causes patient to hold his head rigid. General condition is becoming poor; excruciating headaches; miserable appearance.

Even though the chills, high remittent fever, the cord-like, painful swelling along the internal jugular, and also the plainly discoverable enlargement of the spleen, all pointed to thrombosis of the sinus with pyæmia, another opening of the skull was attempted on August 28th. A new portion of the bone was removed, forming a right angle with the previous incision; a large amount of bone was removed from the canal which led into the interior of the skull and apparently into a cavity existing between the dura and bone; this was accomplished by

means of Luer's chisel-forceps, so much bone being removed that the opening finally assumed the size of a silver dollar. The posterior margin of the trephined opening was distant 6 cm. from the external auditory canal, and the upper limit was 3 cm. above this canal. Very little hemorrhage in general, but rather active from the emissaries at the parieto-mastoid suture. The dura, apparently very much thickened and weakly pulsating, presented a grayish-yellow, fibrino-purulent layer on its surface. The operation, which had evidently exposed the transverse sinus, had apparently no immediate influence upon the temperature; but the chills did not occur again. The increase in the size of the spleen and the swelling in the neck gradually diminished. The discharge from the ear gradually ceased and hearing improved. The evening elevations of temperature continued until the beginning of October, and then they also gradually disappeared. October 22d, the day of patient's discharge, he had recovered entirely. The operation-wound was entirely healed.

An inflammatory process may also travel from the petrous portion of the temporal bone to the interior of the cranium by means of the nerves lying along the base of the skull. Above, this has been referred to in Macewen's second case. Just as the oculo-motorius was affected in this case, the abducens may be involved in others. Cases have been reported, for example by Styx,[51] in which fever, headache, vomiting, and constipation occurred in the course of a chronic catarrh of the middle ear; and then, in the third week, paralysis of the abducens and optic neuritis of the same side were added. If the inflammation extends from the deeper parts of the ear—from the labyrinth and along the carotid—the nerves lying along the wall of the cavernous sinus may be involved. With the liberation of the secretion from the middle ear the affection of the abducens and of the optic nerve disappeared in Styx's cases.

The route by which suppuration advances in the interior of the skull is along the bone, veins, and nerves. It has already been mentioned above that in abscesses of the cerebellum, the pus collects at first between the dura mater and the bone in the posterior fossa of the skull. Among others, Rothholz[51] describes a case of cerebellar abscess occurring with chronic purulent inflammation of the middle ear; in this case, a connected chain of suppurations could be traced from the tym-

panum to the posterior fossa of the skull along the facial nerve, and from this point in the pia mater to the cerebellum and the abscesses in its interior.

Suppurating processes occurring without anatomical connection, when they are found near a diseased ear, as subperiosteal abscess or as caries of the skull, should be regarded as manifestations of a multiple affection of the bone rather than as a continuous disease. This is often the case in tuberculosis of the roof of the skull. During the preceding months we have had several of such cases at the clinic; cases in which the posterior inferior angle of the parietal bone was affected by the tuberculous process causing an infiltration of the cheesy mass throughout the entire thickness of the bone. Though not found in these cases, others representing perforating tuberculous ostitis of the bones of the skull have presented a layer of spongy granulations beneath the diseased bone; this layer of granulations extended far beyond the area of diseased bone; in other cases of this kind, a considerable collection of pus was found. In one of these cases the occurrence of a circumscribed swelling over the occipital bone was preceded by headaches and frequent vomiting; the swelling consisted of a subperiosteal abscess which communicated with another abscess situated between the dura and bone, by means of an opening in the tuberculous area of the bone. Evidently this inner collection of pus had been more extensive at the time when the patient complained of headaches and vomiting, and had subsequently diminished in size upon working through the bone and spreading into the soft external parts of the skull. Just as pus may collect beneath these tuberculous areas of the cavity of the cranium, it may also occur in like manner beneath the affected walls of the mastoid portion of the temporal bone.

I have just had a case under treatment in which, besides a tuberculous sequestrum chiselled out of the mastoid process by me, a fistulous passage leading up to this point, there were two other spots on the opposite parietal bone and on the frontal bone affected with perforating, cheesy osteitis of usual small, circumscribed extent. After such experiences we will be apt to regard the following history of a case as one of multiple tubercular osteitis of the skull. I treated the case with Dr. Trautmann, to whose kindness I am indebted for the following notes:

The patient was a boy eight years old. January 17th he became ill with pain accompanying a profuse discharge of pus from the right ear. Two weeks later, a swelling appeared over the occipital region, also constant high fever, frequent vomiting, violent headaches. For some years past he had had considerable swelling of the lymphatic glands on both sides of the neck; some of these had been removed by a former operation on the left side. Upon admission to Dr. Trautmann's clinic, March 7th, the patient had become emaciated to a skeleton; he was anæmic and had high fever every night; pulse frequent and intermittent; scarcely any appetite; vomiting from time to time. On the right side the softened glands were incised in front and behind the sterno-mastoid. Very marked fulness of the veins on the left side of the face and skull. Examination of the ears showed that of left side to be normal. On the right side, bony conduction preserved; whisper can be heard; fœtid, thin pus from the canal. Upon syringing, the water escapes into the throat and mouth. Large perforation in the anterior portion of the drum-membrane, through which air passes easily. March 22d, severe chill with elevation of temperature to 103.5°. Opening of the mastoid process; the dura was exposed on account of extraordinarily small size of the mastoid. Extensive venous hemorrhage from the soft parts and bone. Pus found in the antrum of the mastoid. The nocturnal elevations of temperature ceased for a few days, but then returned. A canula was inserted in the opening in the mastoid. Each time that this was removed, considerable pus came away; so that the fever which still persisted, sometimes in the morning, sometimes in the evening, was attributed to retention of pus in the petrous portions of the temporal bone. April 26th, an abscess situated behind the upper third of the sterno-mastoid and communicating with the wound in the mastoid, was opened. May 25th, a fluctuating spot over the angle of the lambdoid suture was found and opened; the underlying bone was rough and carious. All the wounds presented very spongy, brownish-red, and easily bleeding granulations. May 29th, I was consulted and scraped out the different wounds so as to apply iodoform dressings. The fever, however, continued. On account of the fever and because retention of pus in the ear could not be demonstrated, I diagnosed an abscess beneath the carious spot in the occiput.

Even though the caries of the occipital bone were not tuberculous, it would still have been advisable to chisel open the diseased cranial wall. Under these circumstances, a fever which almost reached 104° in the evening and fell to normal in the morning, pointed almost absolutely to a collection of pus unprovided with an opening or canal of exit. On this account, I chiselled away the diseased portion comprising an area 4 cm. square; the inner margin of the opening corresponded to the middle line. During this operation also there was hemorrhage from all the small and the very smallest vessels even, so that much time was consumed in tying them. The dura did not pulsate; the value given this sign is well known. But it was discolored and looked gangrenous. I divided it along the outer margin of the opening so as to avoid the longitudinal sinus; there was an immediate escape of considerable pus. A sound could be introduced to the depth of more than 3 cm.; a drainage-tube was introduced a like distance and left there. The fever ceased as a result of the operation. Ten days later, June 26th, on syringing through the opening in the bone, the stream of water came through the drainage-tube which had been placed in the abscess of the occipital region. This experiment was repeated several times and also upon succeeding days. The route which the pus took from the ear to the dura on the opposite side of the longitudinal sinus I will not dwell upon here. The marked dilatation of the veins of the skull and face on the right side, the side of the diseased ear, would direct attention to a thrombus of the sinus. However, extension through the internal jugular was rendered improbable by the absence of tenderness along its course, and of the formation of a hard cord. In considering a thrombus, we might imagine that the excitant to suppuration had travelled to the vena magna Galeni and then to the side of the longitudinal sinus, and had produced an abscess here. The route must have been an open one, for only in this way can be explained how the fluid introduced into the right mastoid process by the point of a syringe could have appeared beneath the dura of the left occipital region. If we leave this communication out of account, we will be compelled to regard the bony affection of the occipital and that of the temporal as two examples of tuberculous osteitis. Above I have already presented an example of a—or rather of two abscesses

of the brain beneath a perforating tuberculosis of the skull in the occipital region; and I regard tuberculosis of the petrous portion of the temporal bone as not uncommonly the cause of chronic otorrhœa. At any rate, the boy was suffering from a local tuberculosis of the lymphatic glands. This led to an extirpation of the chain of glands on the left side after the patient had been discharged as fully cured July 20th, and had returned to Stettin beginning of August. About the middle of September, my colleague, Dr. Schmid, informed me that the patient was well and strong, but that the wound in the occipital region had not yet fully healed. He had been compelled to destroy the spongy, exuberant granulations repeatedly; the suppuration of the middle ear had ceased; the canula which had been left in the mastoid process for a long time without any evil results had been removed some time ago. However, the improvement did not last a long time; Schmid found a new abscess on the right side of the skull, necessitating another operation of chiselling. The boy died during February, 1888. The autopsy was limited to an examination of the skull and was witnessed by Dr. Schmid. This examination showed that the roof of the skull was perforated in several places—the right mastoid process, the parietal region, and a point the width of three fingers behind and above the right mastoid process. At these spots there was an extraordinarily dense adhesion between the membranes of the brain and the bone. There were also cheesy areas in the neighborhood of these affected points. The cerebrum itself was intact. The sinuses, especially the longitudinal sinus, were filled with thrombi, partly firm, partly brittle. The right temporal bone was removed; it was free from pus.

In the face of such a grand series of successfully located and successfully operated abscesses of the brain, the difficulties of diagnosis will no longer be considered insurmountable. This is not intended to imply that we must diagnosticate every case of abscess, but only that we should do so more often than is done at present. I would restrict the operation to those cases in which diagnosis is possible—*i.e.*, those cases which present the symptoms indicating deep-seated abscess of the brain which I have endeavored to enumerate and describe in the preceding pages. Who acts differently will not escape the disagreeable conviction of having undertaken a great and weighty proce-

dure unnecessarily. I come back to that which I said in the introduction to these pages, that only a limitation to certain well-considered and selected cases will favor the progress of brain surgery, while an extension to uncertain and doubtful provinces will restrict and interfere with this advancement. In a word, I advise the operation only in the so-called typical cases, and above all, when the etiological points, already mentioned quite often, correspond. I myself have found out how important this very point is.

Prof. Wernicke, whose knowledge and experience in the line of diseases of the brain are far in excess of my own, called upon me to perform an operation upon a female patient of forty-five, who suffered in all probability, according to his belief, from an abscess of the brain. Unfortunately I no longer possess the very interesting history of the case. This much, however, is certain, that for months the woman had suffered from a febrile disease with severe brain symptoms, such as headache, vertigo, delirium, and great restlessness—a disease which her family physician had positively differentiated from typhoid fever on account of the absence of intestinal disturbances. After this she became well and remained so for some time until, with persistent and violent headaches, well-marked and certain hemiopia developed, followed by pressure symptoms which increased constantly in severity. The main guide in the diagnosis was the former illness, which on account of the fever and brain symptoms could easily be taken for the initial acute stage of an abscess of the brain. Notwithstanding this, I was not convinced of the existence of an abscess, and hence did not desire to operate. A definite etiology was lacking; and in addition, it was certain that the patient had not received a wound of the head nor had she suffered from any ulceration of the head or any disease of the ear, pleura, or lungs. She died not many days afterward. The record taken in the post-mortem room of my clinic reads as follows: At the base of the brain a gray mass presents itself, which is prominent on the left side at the boundary between the frontal and temporal lobes and extends almost to the median line. In removing the brain the end of the temporal lobe fell off; it is permeated by the tumor mass which is adherent to the bone in this situation. Examination of the base shows that the tumor mass has extended into the spongy sella turcica, be-

tween the pyramid of the temporal bone and the sphenoid and into the sphenoidal fissure. On opening the ventricles, the right lateral ventricle is found markedly dilated and the region between the optic thalamus and the adjacent white layer is swollen. An incision here comes upon the tumor mass which involves the layer of white substance extending from the outer region of the optic thalamus toward the occipital lobes. From this point the principal part of the tumor extends through the thalamus, the posterior portion of the lenticular nucleus, and the posterior region of the internal capsule. One prolongation passes to the end of the temporal lobe. Another passes from the optic thalamus to the base of the left crus cerebri. The left optic tract cannot be separated and the left oculo-motorius seems to be lost in the tumor mass. Upon microscopical examination, the tumor is recognized as an infiltrated sarcoma with cysts due to softening.

I have repeated the contents of the report so fully, because in the chapter on the operative treatment of tumors of the brain I shall repeatedly emphasize the impossibility of operating in cases of infiltrated tumors of the brain; not only because they lack sharply-defined limits, but because they have prolongations and extensions which pass here and there in an irregular manner, as in the case just described.

The simple manner in which we can bound that portion of the skull within which the convolutions of the temporal lobes may be reached by chisel or trephine has already been given. Where we have reason to believe pus exists along the transverse sinus and in the posterior fossa of the skull, it will be well to make the centre of the proposed opening in the skull correspond to the posterior boundary line of the field of operation just alluded to. It is always advisable in these cases to make a comparatively large flap, consisting of skin and soft parts and including also the periosteum, which should be allowed to adhere to this flap, the convexity of which should extend downward to the outer surface of the mastoid process. In this way it is possible to expose the whole region of the postero-inferior angle of the parietal bone, so that the junction of the sutures with the occipital bone and the mastoid portion of the temporal bone can be seen. Besides the advantage of better guidance, there is also that of exposing a large emissary vein which is situated, as a rule, some-

what in front of and below the so-called "asterion"—*i.e.*, the point of junction of the three sutures (lambdoid, parieto-mastoid, and occipito-mastoid). If a drop of pus appears next to this vein or is found beneath the periosteum, the fact that suppuration has extended toward the posterior fossæ is made certain. In cases in which trephining is done further back, if we first chisel open the portion lying in front of our posterior boundary line of the temporal lobe—*i.e.*, in front of the vertical line extending from the posterior margin of the mastoid to the parietal suture—we need not fear wounding the transverse sinus, since we will keep in front of it and gain the cranial cavity above the anterior superior surface of the pyramid, where the latter spreads out laterally upon the squamous portion of the temporal bone. If we have made a sufficiently large opening in this situation we can separate the dura posteriorly by means of a broad, blunt elevator or raspatory. In this way we reach the angle between the two upper surfaces of the pyramid—*i.e.*, the attachment of the tentorium in which the superior petrosal sinus is inclosed. If the suppuration has extended along the latter to the transverse sinus, we could also reach it from this point. In cases in which the pus has escaped from the emissary at the parieto-mastoid or the occipito-mastoid suture, however, we would endeavor to reach the groove in which the transverse sinus lies, by carefully chiselling and breaking away the upper part of the mastoid portion of the temporal bone. With some care we can avoid wounding the transverse sinus while exposing it, by chiselling obliquely flat, layer by layer. As soon as it is exposed at any part of its course, it should be separated from the bone by the elevator before proceeding to chisel away the splinters of bone.

Thus the sinus may be exposed, and, as has already been mentioned, this has been attended with surprisingly successful results even when thrombosis had already taken place.

It is not a question of finding every abscess in the hemispheres of the cerebrum or cerebellum, under every circumstance, but of not allowing those abscesses which clinical experience of the present time enables us to diagnosticate, to remain unopened. Even in olden times, traumatic abscess was suspected with greater or lesser probability, but was not found because the dura only was incised, the operator not daring to enter more deeply into the brain. Quesney and

Lapeyronie desired to seek further for the pus in such cases, by means of an exploratory puncture; but they could not conquer the prejudice of the times concerning the unapproachability of the "organ of the soul." Dupuytren was the first to plunge his bistoury into the brain so as to allow the pus to escape. However, his patient died several days after the oncotomy, and this may have deterred surgeons from the use of the knife for such a long time, no matter how much the diagnoses of the great masters of all places were admired and praised. Until the most recent times, puncture with an exploratory or aspirator needle was preferred to incision. This procedure is, however, unreliable and uncertain, independent of a result of an energetic aspiration which must not be lost sight of. Beck states that this once caused a hemorrhage into the lateral and fourth ventricles. Cases in which the operator's experiences were similar to mine described in a former observation are quite numerous; the punctures missed the abscess, which at the autopsy was found close to the exposed cortex of the brain (cf. also Kappeler, "Correspondenzbl. d. Schweizer Aerzte," 1876, S. 106, and Schönborn, "Deutsche Zeitschrift für Chirurgie," 1880, Bd. 13, S. 381).

At times, the exploratory needle might be substituted for the knife. The more reliable the diagnosis is, the less dangerous is the remedy. Two experiences have made us hesitate less in attacking the brain nowadays than did Astley Cooper, who expressed his fear of even extracting loose fragments of fractured bone from the cortex. In the first place, we have become accustomed to remove the débris of brain substance from the depth of complicated fractures of the skull in the interests of better primary disinfection of the wound and so as to thoroughly cleanse the wound. The more exact and careful we were in this procedure, the better would be the healing and the functional results. In addition, physiology, especially that of the cortex, has given us information regarding the great limits of the principle of substituting functions which exist in the structure and arrangement of the brain, and that every sensory and motor province of the body possesses a multiple representation in the central organ. Hundreds of experiments upon the cortex of the brain of animals have demonstrated how any loss which follows the destruction of connecting elementary parts is neutralized and replaced

by the substitution of other elements. The second factor which justifies incision into the brain is based entirely upon the experiments just mentioned. Of all the deductions as a result of operating upon the brain of dogs and apes, that which refers to the indifferent effect of a deep incision into the white substance of the hemispheres, is of especial importance for our purpose. This procedure is not followed by any symptoms, differing in this respect from the experiments of excising a portion of the cortex or of extirpating a section of the gray matter of the surface.

Besides the disintegration of numerous bundles of fibres, the hemorrhage was also feared in cutting into the brain. Hemorrhage from one of the blood-vessels of the brain was associated with the idea of a large, hemorrhagic, so-called apoplectic collection in the interior of the organ. But the excavation and disintegration of the substance of the brain supposed to be produced by incision into the brain are improbable, because the blood would simply make its way to the exterior by means of the open wound. The greatest portion of the hemorrhage in the method of procedure recommended, if not deeper than 4 to 5 cm. and if in front of the roof of the lateral ventricle, is due to wounding the blood-vessels of the pia, and not from those of the substance of the brain. Finally, the use of the provisional and temporary iodoform tampon, which I employ most frequently [53] in bleeding injuries to the brain in the treatment of complicated fractures, gives us an excellent remedy in definitely checking the hemorrhage.

The technique of the opening of an abscess of the brain will no longer deter us from undertaking the operation at present. It is the diagnosis alone from which we must expect everything—*i.e.*, progress in treatment and cure. This must be brought to a certain degree of probability before surgical action can assert itself. This is necessary so that our attack will not resemble the groping of the blind. If, however, the diagnosis is based upon points which we have described as the points upon which all depends, the operation may be risked even in cases in which a mere probability exists. As in all things, there is a point at which diagnostic deliberation ceases and the treatment of the physician begins. The fact that we can accomplish so much more now than formerly in these very cases is evidence of our advanced art. We can proceed be-

yond the diameter of the crown of the trephine which formerly represented the limits. With the chisel we open the skull as much and as extensively as is necessary for our guidance, and then we substitute the broad blade of the knife for the narrow puncture of the needle and the trocar. If we have made a mistake, we are capable of controlling the dangers of our attack and are also usually able to pass safely over them. I cannot take the position that the opening of the cavities of the dura and of the peritoneum are proceedings of no importance. As long as we do not believe in an absolute infallibility in our operations and dressings, we will have to confess that mistakes can be made in our antiseptic procedures, and that these are often made in the very cases in which we believed ourselves most certain. The large material of the great city in which we live should warn us to be strict in discipline and in self-control. I can only regard exploratory trephining and incision of the brain as justifiable in those cases which present the conditions which our investigation has already developed and made certain.

Through the anatomical investigation in Gussenbauer's, my own, and Macewen's cases, the healing of an abscess of the brain which had been opened extensively was demonstrated. This result was not changed by subsequent disturbances or sequelæ. Naturally, in cases in which nerve-tracts have been destroyed and communications interrupted, secondary degeneration may affect certain collections of fibres which have become separated from their centres, and there may also be connective-tissue degeneration of the brain, extending in a radiating and diffuse manner far beyond the seat of the cicatrix. This is possible with every cicatrix in the brain, though it is not necessary. On the other hand, large and extensive cicatrices in the brain have often been found when not expected in the autopsies of persons who have enjoyed a long life in the best possible mental and bodily condition.

The disease which would be most apt to follow a cicatrix in the brain such as a healed abscess would produce is epilepsy. As a matter of fact, this caused a fatal result in the case of Gussenbauer, but in that of Fenger it seemed to occasion an improvement or cessation.

I cannot close the chapter on the treatment of abscesses of the brain without considering the prophylactic treatment. In

this connection I have already spoken of our endeavors to cause a rapid healing of complicated fractures of the skull, without suppuration. The smoothing and the better arrangement of the fractured portion is of the same importance in this connection as the plan once proposed by me of covering the fracture by a plastic disposition of the skin. If the suppuration is completely or almost completely avoided, subsequent abscess of the brain need not be feared. This experience is a warning to those who are intrusted with the treatment of chronic otorrhœa. Modern otology may certainly boast of nice results in this direction. I will only refer to the works of Loewenberg [54] and Kessel [55] and also to the great importance of Schwartze's operation of the mastoid process, since chiselling open the cells of the mastoid process and the antrum of the mastoid have taken the place of Wilde's incision.[56] This does not conclude the operative treatment of chronic suppuration of the ear, however. There is no doubt that in the majority of cases, the careful syringing of the external canal will be successful—*i.e.*, will lead to a permanent cure. But where this healing is protracted, where the drum-membrane has become destroyed and the ossicles have been lost and the suppuration has spread to the cells of the mastoid process and has attacked the bone, more could be done than merely chiselling open the mastoid process and syringing through modern antiseptic solutions. It becomes our duty to give the pus free and constant exit. This is accomplished by drainage of the abscess cavity and rest. Syringing and irrigation may be the means of carrying and pressing particles of pus, substances which produce inflammation, from the collection of pus to neighboring walls of the brain which are softened though not yet suppurating. The stream of carbolic or boric acid solution becomes a means of propagation of the inflammation in this manner. How often I have seen simple gunshot wounds of the soft parts which would certainly have healed beneath the scab if left to themselves, become inflamed on the fifth or sixth day because they had become the object of activity of a physician armed with an irrigator or syringe. I have seen more extensive and forcible syringing in modern aural therapeutics than I would be able to answer for if applied to a wide, gaping wound of the skin!

Most recently, Küster [57] also expressed this view in connec-

tion with the presentation of my case of successful operation in a patient who had had deep abscess in the temporal lobe, at the session of the Berlin Medical Society; he also referred to a long series of personal observations in which he had succeeded in curing the suppuration by a wider opening of the suppurating cavity. As early as 1879 I removed a bullet which had become fastened in the deep portion of the bony auditory canal and had excited acute suppuration with high fever, headaches, a typhoid condition and vomiting, by chiselling away the upper and posterior wall of the auditory canal. Between the upper wall of the external auditory canal and the inferior and lateral boundaries of the middle fossa of the skull there is a pretty strong layer of bone into which the chisel may penetrate to the bony circumference of the drum-membrane without opening the cranial cavity. We can convince ourselves of this by a series of sections with the saw parallel to the base of the skull made above the external auditory canal. The upper boundary is formed by a prominence produced by the transverse root of the zygomatic process, a prominence which extends beyond the outer canal to the external surface of the mastoid process. An incision down to the outer surface of the squamous and mastoid portions of the temporal bone is made around the auricle; it is crescentic, concentric to the antihelix, but at least 2 cm. distant. If the posterior auricular or its branches are wounded, these are tied. Then the periosteum above and behind is separated and pushed back from the circumference of the outer canal so that this is entirely exposed. Then the narrow, sharp chisel is applied immediately beneath the edge of the root of the zygomatic process just mentioned, and then, as is mentioned above, layer after layer is chiselled away so that this instrument passes backward to the mastoid process; from the latter point it advances in an oblique direction against the pars tympanica, forming the posterior wall of the outer canal. We guard against going too deeply and against wounding the facial nerve in the Fallopian canal by the oblique position of the chisel and by removing the fragments of bone through elevation of the instrument driven into the bone in a flat manner. By removing the upper and posterior wall of the canal in this manner, we can secure a funnel-shaped opening which will allow the finger to be introduced into the tympanum so

as to remove the remains of the ossicles, the rigid granulations, and the cheesy, stagnant pus with the sharp spoon. In exactly this manner I operated upon the case already reported on December 16th of last year; the otorrhœa in this case improved during the treatment of the trephine wound, though suppuration still continued. After I had cured his abscess of the brain, I was naturally anxious to cure the disease of the ear which undoubtedly had been the cause of the brain affection. Therefore I exposed the external auditory canal and chiselled away the upper and posterior wall, and also made a wide opening into the mastoid cells in the manner which I have just described. The wound was filled with iodoform gauze and this tampon was allowed to remain two days. Then a drainage-tube was introduced into the scraped-out tympanum; this was constantly being replaced by a finer one as the wound behind the ear gradually filled up with more and more granulations. Finally, on the 23d of January, it was removed entirely. January 30th, when I again presented this patient to the Berlin Medical Society, the wound of December 16th had also healed and the suppuration had been checked; the cotton which the patient had had in his ear for twenty-four hours was dry.

CHAPTER III.

THE OPERATIVE TREATMENT OF TUMORS OF THE BRAIN.

Were I only to follow the impressions made by consultations and by discussions in medical societies and circles, the question of the operative treatment of tumors of the brain would seem to present itself more frequently than that of opening cerebral abscesses; though I believe that the surgery of brain tumors offers less prospects of success and consequently has a less brilliant future than that of endocranial suppurations.

It cannot be my object here to teach the diagnosis of cerebral tumors. This has been fully done in Wernicke's excellent text-book on diseases of the brain. I need only speak of the great demands made upon the surgeon by diagnosis. He who is to operate upon a cerebral tumor must know more than the situation of the tumor, for he must also be aware whether it is possible to enucleate it. It is well known that tumors of the brain appear in two forms: 1, Circumscribed and surrounded by its own connective-tissue capsule, and 2, infiltrated, without any recognizable boundary between it and the beginning of healthy brain substance. As a rule, there is a wide circle around the latter in which the brain is in a condition of red and white softening. The first group displaces the cerebral tissues, the second destroys them and occupies the destroyed portion. The first can be shelled out either with a blunt or a sharp instrument; this cannot be done in the case of the second group. In order to remove and to extirpate them, we would have to—as in cases of carcinoma of the face and breast—carry our incision a considerable distance into healthy tissues; that is, we would have to remove large sections of the organ. Such removal of a portion of the brain is only possible in two situations: in the frontal and in the occipital extremities. In all other situations, the tumors would have to be very small and the infiltration slight, in order that they could be removed in this manner.

The tumors which spring from the bone—that is, from the roof of the skull—will only receive passing mention. The extensive but successful operations which have been performed for their removal are the results of efforts to lead the way to operations upon the interior of the skull, by first making large openings in the roof of the skull. For some time past, numerous examples have shown that a large part of the walls of the skull may be broken through and taken away without interfering with life or with the functions of the brain. Carcinoma of the integument of the forehead has been the most frequent indication for removal of a large piece of the frontal bone. I myself have operated successfully in eight or ten cases in this manner. In one of my cases [58] a third of the frontal bone was removed and the large defect in the skin and bone was covered by an immediate plastic operation, a procedure which I had adopted in analogous cases with excellent results. The patient referred to, a native of Würzburg, was examined three years after the operation, and was not only free from a return of the disease, but was in the enjoyment of perfect health. The absence of bone upon the roof of his skull did not annoy him in the slightest. The cases which Heineke [59] and recently Gussenbauer [60] have collected are even more important and more brilliant. In the case of von Langenbeck [61] there existed an irregular sarcoma of the parietal region of the size of a fist; after its extirpation there remained an opening 8 cm. in diameter; healing took place without interruption. In Macewen's [62] and Gussenbauer's cases the defects were even larger. In the latter case, the entire right half of the frontal bone, with the supraorbital ridge and a small piece of the great wing of the sphenoid and of the squamous portion of the temporal bone, were removed. Recovery followed without rise of temperature or any brain symptoms, and at the end of two years the man was perfectly well and strong. The opening which remained upon the right side of the forehead was closed in part by bony tissue, owing to new formation of bone. The operation for carcinoma of the frontal bone necessitated the removal of large portions of the dura in von Langenbeck's and in my own cases; Küster [63] was compelled to remove the dura and also a portion of the superior longitudinal sinus, which had previously been tied, in an operation for recurring sarcoma.

If in these cases it was made evident that large pieces of

the bony roof of the skull could be removed without ill effects, it seemed natural to ask whether, besides being able to resect large portions of the bone and of the dura, it was not also possible to remove cerebral tumors. We must begin by knowing which cerebral tumors are suitable for operative interference in general, and how such can be recognized.

In answer to the first of these questions, Hale White[64] has contributed an important paper, in which he examined 100 cases of cerebral tumors found at autopsies at Guy's Hospital during the years 1872–84, with reference to the possibility of operative interference. In his report we notice first the relative frequency of cerebral tumors, for in every 59 autopsies one cerebral tumor was found. The 100 tumors represented tubercular tumors, 45; gliomata, 24; sarcomata, 10; glio-sarcomata, 2; carcinomata, 5; cysts, 4; lymphoma, 1; myxoma, 1; gummata, 5; and of uncertain nature, 3.

The syphilitic new formations of the brain, the gumma or the syphiloma, occur quite often in the form of a tumor and are most frequently found on the periphery of the brain, in the dura, or in the subarachnoid space, thence extending to the brain substance. They are therefore accessible, and on account of their definite form, just as in other gummata—*e.g.*, those of the muscles—they may be extirpated by scooping them out. If the gummata are situated in the motor region, then, as is the case with other tumors of this region, we are enabled to diagnose them: in fact, the symptoms are very evident; and owing to the fact that the disease is not an uncommon one, the symptoms of this variety of tumors are well known. After all sorts of general symptoms have manifested themselves, such as psychical disturbances, exacerbating headaches, and sleeplessness at night, the severe symptoms suddenly appear, generally in the form of epileptic convulsions. It is well known that in some cases the paroxysms follow each other so rapidly that an epileptic state which endangers life appears to be present, and we may regard the removal of the gumma as an *indicatio vitalis*.

Independent of the fact that in this condition it is seldom possible to obtain a sufficient number of diagnostic points to decide upon the seat of the new formation, the rapid production of mercury saturation, as in a case which I have recently seen, relieves the dangerous symptoms immediately.

There is scarcely any field in which the energetic internal administration of the preparations of mercury or maximum doses of iodide of potassium produces so rapid and certain a cure as is accomplished when these drugs are administered in cases of cerebral syphilis. It would be inexcusable to begin with the operative before having tried the medicinal treatment. If the latter is employed and continued, a cure is effected in most cases of gummata of the cortex. The question of operation could only be raised after this plan of treatment had been employd continuously and the severe symptoms did not disappear. But in these cases the question arises as to whether a gumma exists at all, or whether only a definite residuum remains thereof—namely, a cicatrix. The extirpation of one cicatrix necessarily leaves another, and it is doubtful whether the patient gains anything, or if he does, how much he gains by the exchange.

The question of separating and removing cicatrices of the cerebral periphery will be considered in the chapter on the operative treatment of epilepsy. But in that connection the case is entirely different, for the cicatrix is one due to traumatism and is of an unfavorable character; we replace the poor one by a better one; whereas in these cases of syphilis we cannot be sure that the new cicatrix will not be as large as the old one was, nor that it will not irritate, stretch, or otherwise interfere with the more or less adjacent portions, just as the old one did.

There have scarcely been any observations in cases of trephining for syphilitic cerebral troubles. Two cases of Macewen's which possibly come under this category will be spoken of further on.

The greatest number of cerebral tumors come under the category of cerebral tuberculosis. The prognosis in this variety is the worst imaginable, and successful internal medication is still a *pium desiderium.* Consequently it would seem plausible to think of adopting an operative plan of treatment in these cases. But unfortunately this plan also meets with a variety of great and often insurmountable difficulties.

Virchow says in his work on tumors (p. 665) that there are a few single cases of large tubercles of the brain which may be regarded as primary cerebral tuberculosis. But in comparison with the large number of cases of primary tuberculosis

in other organs, that of cerebral tuberculosis is a rarity and almost an exception. In White's 45 cases, besides the brain, other organs were affected as follows: In 5 cases, only 1 other organ; in 4 cases, 2 others; and in the remaining cases, 3, 4, 5, 6, and 7 different organs and tissues. If we confine the operation for local tuberculosis to those diseases—in which the infection has not become general—that is, to those cases where, at the most, only two or three organs are affected—there were 9 of the 45 cases which could have been called operative cases.

Of these 9 cases, 1 had to be dropped from the list, on account of the multiplicity of the nodules in the brain, and another, owing to its situation in the pons and out of reach of the knife of the surgeon. Consequently there remain only 7 cases. But of these, 4 must be left out of consideration, 1 because of an associated tubercular basilar meningitis, and 3 on account of the diffuse character of the tubercular masses. Of the 3 which might have been operated upon, being accessible, we learn from White's tables the following. The first case was a tubercular conglomeration situated in about the central portion of the left hemisphere, and with it there was associated a tuberculosis of the peritoneum and Fallopian tubes; in the second case there existed a nodule the size of a walnut in the central cleft of the fissure of Rolando, and also tuberculosis of the lungs and intestines; in the third case there was a nodule in the left hemisphere of the cerebellum, with tuberculosis of the mesenteric glands. Taking for granted that a cerebral tuberculosis existed near the surface of the brain, and owing to the small size of the tubercle was accessible to operation, as in the three cases just mentioned, the question would still not be settled, for the case which seemed most favorable for operation might be accompanied by tuberculosis of the lungs, intestines, and peritoneum. From these facts, it becomes evident that the reduction must still be continued, and thus we find that only one of White's cases was really an operative one, namely, the third of the last three just described; for it is possible to believe that the affection of the mesenteric glands might disappear. In this case the nodule was situated in the left hemisphere of the cerebellum.

I do not believe that I have been too extreme in my limitation of the cases, for another difficulty arises besides those already mentioned. The manner in which the cheesy masses

are imbedded in the cerebral tissue prevents our removing them carefully and completely with a sharp spoon, as is done in scraping away tubercular skin and bone tumors. Our operation is therefore incomplete, and consequently the disease returns in a short time. Furthermore, it is also possible that as a result of the operation we may get worse results: we may induce the dissemination of the process over the pia —the formation of a tubercular meningitis.

The methods of diagnosing a conglomeration of tubercle in the brain are the same as those used in the diagnosis of other cerebral tumors. Consequently we must think of a definite point in the brain—and here we can readily understand that we can only refer to the motor region—as the seat of the tumor. In the second place, we consider the nature of the tumor, whether it be a sarcoma or a glioma, a syphiloma or a tuberculous nodule. We diagnose an endocranial tumor having a position making it accessible to the knife; that is, we decide that the tumor can be operated upon. But after we expose the field of operation, we find a fresh conglomeration of tubercles, or a caseous, degenerated nodule. Then naturally we proceed and attempt to remove the diseased mass.

In spite of all obstacles, the extirpation of cerebral tubercles has not only been performed, but the results have been most satisfactory. If the cases that have been operated upon up to the present time show that the problem may be solved, they also prove the correctness of the statements made above.

The operation for localized cerebral tuberculosis has been performed four times. Of these four cases, two died shortly after the operation and two recovered. A cerebral tumor was diagnosticated in both of the cases that recovered, but the nature and consistence could not be definitely made out, though the seat was accurately diagnosed.

The first case was Horsley's. It is the same case which he presented at the meeting of the British Medical Association and in reference to which Charcot and others very properly congratulated him, as did also the prominent men of his own country.

A man twenty years old had frequently suffered with attacks of pleurisy since his fifteenth year; he was first taken ill in January, 1884; he had clonic spasms of the thumb and

index finger of the left hand, occurring about twice a day. His first severe convulsion came on in March. He fell down, the convulsion beginning in the right arm and extending to other portions. A second seizure of the same character occurred in January, 1885. From this time until August, 1885, he only had slight spasms; then, however, a series of severe paroxysms once or twice a week, these continuing until December; the character of the paroxysms was always the same. They began with clonic spasms in the left thumb and index finger, then the muscles of the wrist became affected, then the elbow and shoulder, and finally the face. Then the patient lost consciousness. His eyes turned toward the left; then there followed, in succession, twitchings of the left upper extremity, of the right upper and finally of the right lower extremity. After the paroxysm there remained a paralysis of the left extremity. When examined in April, it was observed that the strength in the left upper extremity was much diminished, although he could make all the various movements. The left thumb was usually rigid and stiff when not in a condition of clonic spasm. The patient complained of severe headaches, which began in the occipital region and extended toward the right parietal region. The entrance of the optic nerve showed no pathological changes. A diagnosis was made of an irritation existing near the border of the inferior and middle third of the ascending central convolution, the seat for the centre which presides over movements of the fingers, especially that of abduction of the thumb. Nothing definite could be made out concerning the nature of the disturbance. Horsley applied a broad trephine over the designated spot on the 22d of June, 1886. A tumor came into view after the dura was raised. In order to obtain a view of the tumor, more bone was removed, comprising all that portion to which the dura was adherent. The tumor extended beyond the surface of the brain and only appeared to be half an inch in breadth. By a free incision into the healthy surrounding tissues, Horsley removed all of the diseased portion, and with it and bordering upon it a bluish-red, discolored zone of gray substance. The new growth extended under the cortex for a considerable distance. The operator convinced himself that the cortical centre for the thumb had positively been removed. Numerous vessels were tied. The wound was im-

mediately closed, drainage, Lister dressing. Five-sixths of the wound healed by primary union by the end of the first week. At the lower end of the skin flap a gangrenous slough came away; but with this exception, there was uninterrupted healing. The day following the operation a paralysis of the inferior branch of the facial nerve developed, as did a paralysis of the left upper extremity including the shoulder. On the 27th of June an examination revealed diminished tactile sensibility, but preservation of sensibility to pain and total loss of muscular sense. By August the patient regained all of these sensations, including the use of the extremity, with the exception of movements of the fingers, which were still absent. In the tumor, surrounded by dense connective tissue, were found two cheesy masses.

In the tables which Horsley published in April, 1887, regarding his operations, he states that later on—in September, October, and November—the convulsions recurred, but were confined to the left arm and shoulder.

The case is as important in respect to diagnosis as it is in regard to the operation; for Horsley overcame the difficulties which were due to the diffuse boundary of the cerebral tubercle by means of incisions which extended into the healthy surrounding tissues, and thus he extirpated the entire diseased region.

In the second case of successful operation of cerebral tubercle, the disease had affected the centre for the great toe, which the operator searched for. The case was one of Macewen's, but I regret that I am unable to state more concerning it than was obtained from the short report which the author gave in the lecture, already referred to, at the annual meeting of the British Medical Association.

The case was one of a girl seven years old, who suffered from very frequent epileptiform attacks, followed by paresis of the muscles which were affected during the paroxysm. At the beginning of the attack, the patient felt a very severe pain in the great toe of the right foot, so severe that she shrieked with pain. This was followed shortly by a tonic spasm with extension of the great toe, this condition generally lasting about five minutes. Occasionally the paroxysm would cease at this point, but more frequently these symptoms were followed by clonic spasms of the right foot, leg, and thigh as

far as the groin, and here they generally ceased. Sometimes the muscles of the trunk, the face, and the upper extremity upon the right side became affected during a paroxysm, and on rare occasions later on it spread to the other side of the body, and then the patient would become unconscious. The strength was markedly diminished in that portion of the body which was affected, but cutaneous sensibility was fully retained.

From these symptoms, a diagnosis was made of cortical disease of relatively limited extent. The peculiar feeling of pain and the fact that it constantly began in the great toe pointed to the upper portion of the central convolutions as the seat of the trouble. The general condition of the patient and her history relating to the health of her parents rendered it probable that the case was one of nodular tubercular disease, which might possibly be multiple.

In operating, the uppermost portion of the anterior central convolution was exposed. This was covered with a few tubercular nodules the size of a barleycorn, which were attached to the vessels over the anterior central convolution. Upon the surface of the brain there was nothing to be seen. When Macewen pushed the central convolutions carefully aside, he found a distinct circumscribed nodule in the upper part of the posterior central convolution, buried entirely in the substance of the brain, which, after the overlying cortical portion had been cut away, proved to be a tubercle the size of a hazelnut and admitting of easy shelling out. As the immediate result of the operation, there was restlessness of the extremities and a trembling affecting the muscles of the right side of the body, especially the right upper and lower extremity. This condition lasted for about one week, and then gradually disappeared. More than a year has passed since then, without any return of paroxysms, and the patient feels perfectly well.

The case was an exceptionally favorable one both as regards diagnosis and in that the size was only as large as a hazelnut, although the presence of small gray nodules along the course of the vessels of the pia would warrant the suspicion of beginning tubercular meningitis. This, however, did not develop, since the patient was perfectly well at the end of a year.

The difficulties attending the operative removal of cerebral tubercles are well illustrated in the cases described by Horsley and Bennett May. Regarding Horsley's case, I am only acquainted with the short extract which he published in his tables in 1887.[65]

The patient, eighteen years old, suffered with gradually increasing headache, vomiting, optic neuritis, increasing weakness in all the limbs, but especially in the left arm and leg; the sphincters were also affected. Furthermore, he had epileptic paroxysms, during which the head and eyes turned toward the right. He had been confined to his bed for one year and was in a most pitiful condition, but throughout this period his mental condition remained unimpaired. On the 17th of December a trephine was applied over the right hemisphere of the cerebellum, and a tubercular mass was removed weighing seven drachms. Listerism with spray was employed. The patient died nineteen hours after the operation. The operator regarded the operation as a *dernier ressort.* The autopsy revealed general tuberculosis of the viscera.

It cannot be denied that had this condition been diagnosed, the operation would not have been attempted. This case certainly confirms all the difficulties of which I have previously spoken.

These are illustrated in a no less forcible manner in Bennett May's operation.

Bennett May[66] was called to attend a patient in whom a tumor of the cerebellum had been diagnosticated. At first, May believed that the affected cerebral portion could scarcely be reached, or if so, only with the greatest difficulty; as a matter of fact, his experience was just the reverse. A boy seven years old was admitted to the hospital on the 19th of July, 1886. In April, 1886, he had suffered with severe frontal headache and had vomited. From that time on his visual powers diminished. The pupils were dilated and irregular, but reacted to changes of light. The eyes showed a conjugate deviation toward the left. The right rectus externus was paralyzed. On both sides there existed severe neuro-retinitis, so that the boy was almost blind. His gait was staggering, and it seemed as if he were going to fall backward and sometimes toward the left. His memory was clear; he spoke sensibly and complained of his loss of sight. Lungs were normal.

Occasionally he vomited. He complained of frontal headache and failed visibly. After a short time he was no longer able to stand. On the 20th of August retraction of the head was observed. On the 8th of October he became totally blind and nystagmus existed. Patellar reflex was absent on the right side. Emaciation was very marked and there existed a condition of collapse. The first incision, which was convex above, exposed the linea arcuata superior, having been carried from one mastoid process to the other. Then the insertions of the muscles were dissected away, almost to the foramen magnum. After the bone had been thoroughly exposed, the trephine was placed upon the centre of a space which was bounded above by the right linea arcuata superior, below by the foramen magnum, and to the inner side by the occipital crest. The dura was exposed for a considerable distance and incised in such a manner that it could be turned back in the form of a square flap.

The surface of the cerebellum appeared to be healthy; but upon palpating it a hard spot was found upon the outer half of the exposed surface. Here an incision was made with a tenotome. The finger which was introduced into the opening discovered a hard mass about an inch below the surface. Its tubercular nature was easily diagnosed by scraping off a piece; then the remainder was removed with the handle of a small teaspoon. The mass was about the size of a pigeon's egg, hard and horny upon the surface and cheesy in the centre. Hemorrhage was slight. No especial disturbances of the brain were observed in the child. The flaps of the dura were then replaced and stitched together. The patient died of shock a few hours after the operation. An autopsy was not granted. Consequently it remained a question how far the tuberculosis had extended into the brain and along its base.

German compilers have repeatedly considered a case operated upon by Macewen [67] as one of cerebral tuberculosis; but the history shows, without a doubt, that it was a case of syphilitic tumor. It is the only example in which a syphiloma of the cortex has been successfully removed.

The woman, twenty-five years old, had previously had syphilis. The left side of her body was paralyzed, a brachiocrural hemiplegia, sensation remaining intact; this condition developed gradually after having been preceded by pricking

pains. Patient had been treated in two different hospitals with antisyphilitic treatment before she came under Macewen's care for operation. I did not consider the facts just mentioned in the short account of the clinical history in the first edition of this work, because I have so often observed that antisyphilitic treatment without result does not justify our discontinuing the treatment in cases of long-standing syphilis. The words, "as the treatment (that is, the treatment of Dr. Wood Smith, etc.), especially that by counter-irritation and prolonged administration of antisyphilitic remedies, had failed to effect the slightest improvement, it was decidedly advisable to have recourse to operative measures," I considered as referring to the treatment which had been previously adopted, and which was discontinued three weeks before the operation, and not as referring to a plan of treatment pursued by Macewen. In this respect I seem to have been in error, for a letter from Macewen's assistant informs me that the former had treated the woman very energetically with antisyphilitic measures, and that, among other means, inunctions were employed until salivation was produced and iodide of potassium given up to 100 grains a day; after this had proven useless in its effect upon the hemiplegia, he proceeded to trephine. After this correction, I have no right to give any but a favorable criticism, in spite of my decision in my first edition, especially since the result proved that the plan of treatment was a justifiable one.

Macewen removed a piece about an inch square from the right parietal bone; then he made a crucial incision into the thickened dura; beneath this, that portion of the surface of the brain corresponding to the middle and upper third of the ascending frontal convolution and the central sulcus was covered with an opaque, yellow, friable mass. When the upper part of the ascending frontal convolution was cut into at the spot where the brain tissue gave a greater amount of resistance, a small quantity of cloudy, brown fluid escaped. The wound was then closed after reinsertion of the pieces of bone which had been cut away; it healed rapidly. At the end of the first week the patient was able to move her fingers and toes, and at the end of two months she could walk very well. In the course of time she regained her former strength in the left extremities almost entirely. At the end of two years her condition was perfectly satisfactory.

If the case be the same as the one to which Macewen refers as No. 6 in his Glasgow report, he says in conclusion, "excepting that the gait is somewhat spastic."

The numerous communications and contributions tending to advance brain surgery must be subjected to a critical examination in order to estimate the importance of trephining for extirpation of new growths in the cortex properly. In the first place, all those cases must be excluded and omitted in which there exists a recent or an old collection of blood and its residue, or a deep depressed fragment of bone, thickening of the meninges, encephalitic degeneration, and cerebral cicatrices. How far we can and should go in regard to the removal of cicatrices will be fully taken up in the chapter on the surgical treatment of epilepsy. These certainly do not belong in the chapter on removal of cerebral tumors, although they are so frequently placed under this heading. In many of the cases, including Macewen's, which I described on page 60 of my first edition, it appears to me that trephining was unnecessary and therefore useless. Although concerning this case (I believe it is the seventh one in his Glasgow report) the operator says: "At the operation, a hemorrhage into the brain was found at this point, with encephalitic affection of the surrounding portions," still I must recall the fact that hemorrhagic masses in the brain, as in muscles, are most readily reabsorbed when they remain subcutaneous and uninterfered with. To expose and to remove them by taking away a button of bone is an inexcusable proceeding and is certainly no advancement in brain surgery.

It is possible in case of uncertainty in diagnosis—and the majority of the diagnoses in these cases are uncertain—for us to meet with an encephalitic mass instead of a tumor; then it is consoling that modern antisepsis protects us from the most serious consequences of our error and also warns us to be more careful in future. It is the same here as everywhere in the realms of modern and even most modern surgery—"*mente prius chirurga agat quam manu armata.*"

If, after these reports of the extirpation of conglomerations of tubercles and of syphilomata of the brain, we proceed with the analysis of White's cases, we must begin by omitting from the list carcinomata, because these were all secondary metastatic tumors. Among the 36 tumors there were glioma

(24), sarcoma (10), and gliosarcoma (2). I will describe them collectively, because, particularly in the case of these tumors, there is so much uncertainty in their nature and they are named so arbitrarily. Thus, for instance, a case of glioma is ascribed to the dura mater, whereas, according to Virchow's views, this form of tumor, which was first described by him, never affects the meninges. Furthermore, the fact that to ten encapsulated tumors there were only eleven infiltrated ones speaks against the correctness of the diagnosis of glioma, for the gliomas in more than nine-tenths of all the cases are infiltrated tumors. If I exclude from these 36 cases 15 diffuse and largely infiltrated ones, then only 18 remain [this discrepancy in arithmetical figures exists in the German edition; the translator did not feel justified in changing the figures]; but of these 14 were not accessible, either because they were multiple and generally secondary, or else on account of their situation in the genu of the corpus callosum, in the pons, etc., so that in only one of them could the question of an operation have come up. Of the four gliomas, or sarcomas, one was situated in the median line of the cerebellum; it was found accidentally at an autopsy on a patient who had died of pyæmia. The second one originated in the dura and produced pressure upon the cerebellum; the third was the size of a walnut and was situated in the frontal lobe; and the fourth was of a considerably larger size and was in the occipital lobe. To these four cases of tumors, which might have been operated upon as far as their anatomical seat was concerned, two tumors must be added from a group concerning the histological nature of which nothing definite was ascertained, and three cases of White's must also be included here. One of these three was not accessible on account of its situation, but the other two could have been found, because one was situated in the left hemisphere of the cerebellum and the other was attached to the dura over the right hemisphere of the cerebrum. Regarding the four cysts, probably hydatids, two were multiple and one was found accidentally in a case of carcinoma of the neck; the fourth could easily have been removed, for it lay completely isolated in the cerebellum. A small, superficial myxoma could also have easily been removed.

If we consider these cases collectively, of the 100 of White's cases of cerebral tumors, only 9 could have been operated

upon, namely, 1 conglomeration of tubercle, 4 gliomata and sarcomata, 2 indefinite new growths, 1 cyst, and 1 myxoma.

The nine per cent of operative cerebral tumors would not represent a small proportion, were it not necessary to reduce the list still further. Not only the anatomical seat and the manner in which the tumor is circumscribed, but also the absolute size of the tumor, influences the possibility or impossibility of an operation; this is decided first of all by the clinical appearances and symptoms. Of what use is it for the pathological anatomist to show us a small and easily isolated tumor in the frontal lobe of the cerebrum, or in the hemisphere of the cerebellum, to prove the possibility of its removal by operation? In the positions just given as examples, these tumors produce no symptoms whatever. Their discovery on the cadaver is an accidental one and not disclosed by any previous indications. It is worthy of notice that of the 9 operative cases of cerebral tumors in White's tables, not less than 5 were situated in a hemisphere of the cerebellum, 1 in the frontal and 1 in the posterior portion of the occipital lobes. I think I can safely take for granted that of the 7 cases, not one gave any special symptoms; in the case of the myxoma, White himself says that, easy as its removal would have been, it was just as impossible to diagnose it, for it produced no symptoms whatever.

However, it is not solely the fact that a cerebral tumor does not produce apparent clinical symptoms; the consideration as to whether a tumor may be considered amenable to operation is governed by whether it gives positive symptoms which show us its situation. An endocranial tumor which has not interfered with the general health of the patient during life does not interest the clinician any more than the general symptoms of the diagnosticated tumors have an operative interest for the surgeon. It is commendable that neuro-pathologists can determine the existence of an intra-cranial new growth with certainty from the general cerebral symptoms, the headache, the vertigo and vomiting, the convulsions, the choked disc, the extreme weakness, the lassitude and the annoying general malaise, and finally the effect upon the sensorium increasing to sleepiness and somnolence. But from all of these symptoms the surgeon obtains no bearings which are of use for an operation. If positive proof as to the seat of the tumor

is wanting on account of absence of direct area symptoms, not even the question of explorative trephining can come up.

The advances which the teachings of the localization of cerebral diseases have made, permit the clinician oftener now than formerly to be in a position enabling him to form more definite and positive conclusions as to the seat of the cerebral tumors. On this account, and because we can open the cavity of the skull with a comparatively small amount of danger, the consideration of the treatment of brain tumors has passed from the clinician on internal medicine to the surgeon.

If it can be demonstrated that the tumor lies in the cortex of the brain, or at least just below it, then, in order that the operation may be possible, we must be certain of two qualities of the new growth: it must not, as has already been said, extend diffusely into a zone of red or reddish-yellow softening, and it must not be too large or too extensive. In the first case we are unable to include and to cut around the border of the soft infiltration; these tumors do not possess the essentials necessary for extirpation—that is, limitation and at least distinguishability from the brain through their consistence; and furthermore, they spread for some distance and in different directions, running through the brain in wonderful prolongations and extending across deep fissures and sulci in strands and bundles. I need only call attention, at this point, to the result of the autopsies given on preceding pages. In the last case, that of a very large tumor, a favorable result from an operation could not have been expected, on account of a rapidly forming cerebral œdema. In regard to the diagnosis as to whether the tumor is infiltrated or circumscribed, we possess just as few points to help us as we possess in regard to the size of the supposed or even the positively recognized tumor. We can say in general that when, besides a few moderately developed, late forming area symptoms, we also have distinct and rapidly forming pressure symptoms, there is a strong probability of the existence of an infiltrated tumor. In reference to the size, we frequently have no indications whatever. We are most able to estimate most readily those in which the tumor grows from the bone into the brain: these tumors usually push their way for a considerable distance into the cavity of the skull before they affect the cortex, even when they are located over the motor region. If we observe that

in the case of a tumor commencing in the roof of the skull we have aphasia, facial paralysis, hemiplegia, or hemianopsia, then we can safely take for granted that that portion of the tumor which lies in the cavity of the skull has assumed quite considerable dimensions.

We must not depreciate the danger of an operation for removal of a large cerebral tumor, even though it is inclosed in a capsule. This danger is due to hemorrhage or cerebral œdema; the latter is intimately associated with the breaking away of large pieces of the skull and with the measures taken to control hemorrhage, especially the hemorrhage from the sinuses.

In order to illustrate these dangers I will relate the histories of two cases. The first is taken from an article by Birdsall [68] and the second from personal experience.

A Polish Jew, forty-two years old, had always been healthy until August, 1885. He denied having had syphilis or any previous injury to his head. At this time, after a sea-bath, he noticed unsteadiness in gait and had a severe attack of vomiting, double vision, and numbness in the right leg, right hand, and shoulder. The diplopia was transitory. He suffered from repeated though not severe attacks of headache; had vertigo. When patient attempted to seize and raise objects, he often failed. On the 7th of October, Seguin first diagnosticated hemianopsia. The muscles of the eye were normal. Both pupils active, the left a trifle wider than the right. On the left side, vertical hemianopsia. No paresis of the face, tongue, or limbs. Patellar reflex normal. Walk somewhat staggering, with decided tendency to turn to the right. On the 7th of November there appeared the first traces of a neuro-retinitis. The external recti of both sides were paretic; again diplopia appeared, this disappearing later. There was drowsiness and difficulty in rising from the chair. A tumor was diagnosticated involving primarily the cuneus and extending to the tentorium. Patient was put on iodide of potassium. From January to July, 1886, the diplopia disappeared. The gait improved somewhat, although on two occasions, while on the street, the patient was unable to move. He staggered now more toward the left. From July till September he was in California. Upon his return, all the disturbances had increased. In October, 1886, the markedly pronounced left

hemianopsia was recognized by Birdsall; also double optic neuritis, most marked on the left side. The left pupil somewhat larger than the right. No diplopia. Hearing, smell, and speech normal. There was no anæsthesia and no analgesia. No paresis of the muscles of the face or of the extremities. The movements were clumsy rather than ataxic. When the patient was asked to touch his nose with his eyes closed he succeeded at times, but more frequently he was wide of the mark, this being particularly noticeable with the left hand. His gait was slow and uncertain. There was no loss of strength. The trouble lay in his inability to control his movements. The patellar reflexes were active and about equal. On account of his disturbance of vision, he frequently ran against objects to his left and he turned his head half-way toward the left. His disturbances of equilibrium increased in an irregular manner. At times he could stand quite well, also sit and walk; at others he would fall forward and toward the right side even when sitting, and he could scarcely lift his feet from the ground.

Some days he would be dull and listless, on others bright and talkative. His intellect was not impaired, his family scarcely noticing any change in his disposition. He never had hallucinations. He slept well. His complaints and being out of humor were due to the disagreeable sensations and difficulty in using his limbs. Lately, the symptoms of cerebral pressure had greatly increased. The diagnosis of a tumor in the cuneus was made on account of the hemianopsia and on account of pressure of this part upon the cerebellum through the tentorium, producing disturbances of equilibrium. After a consultation of experienced specialists, the operation was performed on the 9th of March, 1887, in the New York Hospital. A flap three by three inches was formed with its base upward. The rounded apex extended downward to the level of the right half of the linea arcuata superior, the base extending from the side of the right parietal bone to a little beyond the left of the median line. Two buttons of bone were removed and the intervening bridge gnawed away with a bone forceps. The defect in the bone extended in the middle as far as the angle between the longitudinal and transverse sinus, and was two and three-quarters inches in length and two and a quarter inches in width. The dura did not pulsate; was of a

deeper hue than normal. It was incised around the border of the bone as far as its inner side; here it was not touched nor turned back, in order to avoid an injury to the sinus. Immediately the tumor rose in the bony opening. It was recognizable at once on account of its rose-red color; it was covered in by thin cellular tissue having a large vein ramifying in it. With the director and edge of a spoon handle a layer of cerebral tissue was separated from the tumor on its outer side. In this direction the enucleation was accomplished to a depth of nearly one inch. It was similarly loosened on the other side. Although the opening in the skull was very large, still it was not large enough to reach the end and the base of the tumor. The tumor was therefore incised, and a portion of its softened, granular, and fatty parenchyma removed. This enabled the forefinger to be passed between the cranium and the tumor, and by its aid, but not without tearing the tumor, it was possible to peel it out from the depths. First the outer and then the inner half were lifted out, the latter being removed from the falx. Inspection of the wound showed that the tumor had been entirely removed, and that its attachment and origin had been the posterior border of the falx. The severe venous hemorrhage the operator tried to control by pressure with sponges. The tumor had proliferated over the falx and pushed it somewhat toward the left, and the tentorium was deeply depressed. The tumor weighed 140 grammes. It measured $3\frac{3}{4} \times 2\frac{1}{4} \times 2\frac{1}{2}$ inches; greatest circumference, $8\frac{1}{2}$ inches; least, 7 inches. When examined later, it was found to be a spindle-celled sarcoma. After the brain had been separated with retractors, two bleeding points were seen, one in the region of the longitudinal sinus and the other apparently an artery from a terminal branch of the posterior cerebral artery. The operator tried to control the hemorrhage by direct compression with iodoform gauze. For this purpose the entire cavity of the wound was filled loosely with strips of the gauze. The dura was then sutured, as was the skin, far enough to allow the piece of gauze to emerge from the lower end of the scalp wound. Besides this, a drainage-tube was pushed under the skin to the opening. Over the wound a peat dressing was placed, which was saturated with iodoform and was sublimated.

Toward the end of the operation the pulse could scarcely

be felt, owing to the patient having lost about twelve ounces of blood. The constant oozing of blood from the soft parts and wound in the bone, in spite of the large number of artery forceps applied, increased the loss of blood and increased the difficulty of examination of the deep portions very considerably.

The limbs were bandaged, heat applied, head lowered, subcutaneous injections of alcohol and counter-irritants to skin and rectum applied; these overcame the great weakness. The pulse became strong and rose to 132. He received milk every two hours.

A short time after this the bandage was saturated with blood and the pulse became feebler. Five hours after the operation the saturation of blood was even more pronounced. Patient was conscious; the pulse was very feeble and small. An injection of salt solution with the addition of sugar, after Landerer's method, was given. Thereupon the patient became more quiet and was able to answer questions put to him, so that a continuation of the hemianopsia could be made out. The constant bleeding necessitated a rapid removal of the dressings. The blood flowed continuously from the drainage-tube. It was impossible to grasp the vessels, because when the trephined surface was exposed, the pulse, which had improved after the infusion, ceased altogether. All that could be done was to place more iodoform gauze over the wound. Again salt solution was injected, but without effect. Nine hours after the operation the patient died. An autopsy was not granted. Under the iodoform gauze an enormous coagulum was found.

The operator blames himself for not having properly controlled the hemorrhage. I always recommend seizing the large arterial vessels and tying them. I would never permit hemorrhage from the thyroid artery, for instance, to be controlled by an antiseptic tampon. I consider it just as incorrect as to tie the main artery after an amputation of the thigh and then to control the hemorrhage from the profunda and perforating arteries by a tampon left in place for a short or long time, as I am sorry to say has been recommended. This method may produce good results at first, but later we get secondary hemorrhage or else an accumulation of blood in the deep portions of the interstitial muscular tissues. We can

cut into the brain for a depth of 2, 3, or even 4 cm. without an artery spurting—in fact, without having scarcely any hemorrhage, but if, in removing the occipital extremity at its under surface, we injure a large branch of the posterior cerebral artery, we should, as demonstrated by the history of the case just cited, seek for the vessel and tie it. I will have more to say in this connection later on, in giving the history of a case which Keen relates. The case terminated favorably, but the patient passed through quite a list of dangers before he finally recovered. The succession of dangers and disturbances incidental to the healing of the wound might all have been avoided had it been properly filled with iodoform gauze. Tamponing with sterilized gauze with iodoform powder rubbed into it acts splendidly in controlling hemorrhage. It lessens the dangers of accidental injuries to the brain or those done in operating to a greater degree than any other antiseptic measure. It is not easy to control hemorrhage from the vessels in the pia in dressing a compound fracture of the skull where an injury of the brain has also taken place. This has been especially noted in the history just related; it showed how difficult it was both to control the hemorrhage and to overcome the disturbing and constant oozing of blood from the meshes of the diploë. The iodoform tampon controls this, but must be applied in a different manner than was done in this case. A crumpled piece of iodoform gauze about 40 cm. square should have been placed into this large cavity; on top of this a second and then a third piece, and so on until these extended above the level of the wound in the skin. The skin flap, if one was made, should have been turned back, and the usual antiseptic dressings should have been placed over the layers of iodoform gauze, which filled and extended beyond the wound. The pieces of the dressing, no matter whether of mull, moss, or wood-shavings, are so hygroscopic that they absorb everything which comes to the surface of the iodoform gauze, and consequently the gauze is kept comparatively dry. If, however, we only place a strip of iodoform gauze into the bottom of the wound and allow it to protrude through an opening in the external wound, this strip which was used as a tampon soon becomes so saturated that it cannot absorb any more, and consequently the blood and the transuded serum accumulate alongside of and behind it.

The portion of the strip which protrudes between the edges of the external wound is also compressed, and consequently any idea of drainage from the depths of the wound is an illusion. This procedure was the worst one imaginable, for even drainage without the addition of iodoform gauze would have been more advisable. They really accomplished just what they wished to avoid: namely, the accumulation of compressing and infiltrating wound products which extended for a considerable distance into the tissues, and which were very prone to decompose in the bottom of the wound. I have observed, in following this plan in cases of amputation, that in removing the strip of iodoform gauze, a large quantity of blood-red fluid came away with it. Therefore, although I have recommended and still do recommend the tampon most highly in cases of injuries to the brain, still, everything depends upon its proper employment and its use so as to act as a compress and to carry off the products, but not so as to block up and obstruct the wound. It is true that this plan often requires the administration of chloroform a second time, so that the gauze may be removed quietly, and that the edges of the wound may be approximated and sewed up; but this objection, if it be one, is more than overbalanced by the possibility of obtaining primary union even if the stitches are put in two, three, or even four days after the operation.

I do not wish, however, to underrate the difficult position in which Birdsall was placed in operating upon this patient. Cutting through a large portion of the brain and through one of the larger sinuses is always a risky proceeding, and when it is impossible to tie and transfix the bleeding points at once, the patient is in imminent danger. To apply the above-described and only useful method of controlling hemorrhage in the deep portions of the brain often meets with insurmountable obstacles. Therefore I believe that when cerebral tumors have attained a certain size, it is better to abandon the operation than to finish it. It will not do to say in reply that the patient cannot live and therefore we are justified to try this extreme method. This extreme must, however, always be a curative one, and when such is not the case, it does not come under the category of surgical therapeutics.

The development of acute and rapidly fatal cerebral œdema is another danger which we have to contend with in operating

on large cerebral tumors. This I discovered in the following case:

Mr. S., a merchant, sixty-six years of age, had noticed for the past twenty years a tumor the size of a walnut, situated upon the head over the left parietal bone; this condition remained unchanged until nine months ago, the spring of 1886. At this time it began to grow rapidly and to change its consistency; from bony hardness it became soft. The employment of iodide of potassium did not retard its growth. The patient, however, had no symptoms until September, 1886. Then he noticed for the first time the sensation of formication on his right hand, this condition constantly growing more marked and the hand becoming numb and losing its strength. At the end of three months—when admitted to my clinic on the 8th of December, 1886—he was unable either to grasp or to hold firmly any article in this hand, not even when he steadied the hand. When his eyes were tied, it was utterly impossible for him either to lift or to hold anything. For the past four weeks his relatives had noticed that he became languid, sleepy, heavy, and miserable, and when talking there apeared to be an interference with the movements of his tongue. Occasionally it would take him a long while to try and find a word, and then was not successful. His memory became poorer, in fact was almost entirely lost. He could no longer say his prayers, and although he had been an excellent mathematician, now he was unable to add two numbers together. When writing he omitted words and substituted wrong words. He had never complained of headache or vertigo.

Upon the left side of the head was found a hemispherical tumor, about three or four fingers in height, which ascended gradually from the skull and was highest about in the region of the parietal eminence. The scalp covering it was somewhat tense, otherwise unchanged and movable. The base of the tumor flattened gradually and extended to the sagittal suture and forward to the coronal suture; below and behind it was more sharply curved than above and in front. The lower portion of the tumor was as hard as bone, whereas the upper, larger portion was softer and had the consistency of moderately tense muscle. The surface was smooth, there being no line of demarcation between the bony part at the base and the softer part above. The tumor was permeated by large

pulsating vessels. When the hand was placed upon it, it felt as if it pulsated as a whole, but more careful palpation showed that the pulsations were conveyed along certain channels only. The tumor could not be reduced in size by pressure, nor did pressure produce any special symptoms. The pulse was not slowed, respirations were regular, temperature was normal. No changes in the fundus of the eye. Sensation was diminished in the right hand; superficial pricks with a pin were scarcely felt and were always incorrectly located; tactile sensibility seemed to have been completely lost. Mobility was retained in all portions of the body, though all the movements of the right hand lacked power, and pressure of the right hand, in comparison with that of the left, was scarcely perceptible. The right facial nerve was paretic; the right angle of the mouth was lower than the left; the eye could only be partly closed.

I diagnosed the case as one of myelogenic sarcoma, which had only recently worked its way through the external layer of the bone. Since the tumor could not be made smaller by pressure, I considered it separated from the cavity of the skull by a thin layer of the vitreous table. I was strengthened in this belief by the fact that just as no pressure symptoms existed, they could not be produced by compressing the tumor. The symptoms of paralysis upon the right side and the disturbance of speech, I attributed to a direct effect upon the cortical substance by the growing tumor. Upon this occasion I discovered, for the first time, that a tumor emanating from the bones of the skull only produces cerebral symptoms after it has grown into the cavity of the skull for a very considerable distance. Small elevations upon the inner surface of the bone produce no cerebral symptoms as a rule.

The fact that an enlargement had existed upon the bone for the past twenty years, induced me to give a comparatively favorable prognosis regarding the operation. I counted upon the existence of a distinct and firm encapsulation of the tumor from the surface of the brain.

On the 10th of December, 1886, after the field of operation had been thoroughly prepared, I cut through the soft tissues over the tumor, making a crucial incision; there I chiselled away the entire left parietal bone along its base, taking with it a piece of the frontal bone. At the vertex I was compelled

to go beyond the median line. The bone was completely perforated as was also the dura, and in its centre lay the tumor. The perforated portion of the bone formed a sort of isthmus; in other words, the tumor was shaped like a sand-glass, a portion of which, about the size of a small apple, grew into the brain and was so firmly united with the brain that I was compelled to remove quite a section of the latter, in extirpating the growth. Among the larger vessels, numerous branches of the meningeal artery, which extended into the tumor, were tied during the operation. In removing the tumor along the median line, I was compelled to extirpate a large piece of the longitudinal sinus. The hemorrhage was considerable, but its control not very difficult. The dressings applied were those already described, using the iodoform tampon over the entire wounded surface. The day following the operation the patient was unconscious, but after this he regained consciousness. At the end of forty-eight hours the iodoform gauze was removed and the wound sewed up, but with drainage. On the third day, the patient again became somnolent, answering questions, however, but otherwise he lay in a listless condition. The pulse became more rapid, the temperature rose to 100.4°. The sopor became deeper, and on the morning of the fifth day after the operation the patient died.

The autopsy revealed very extensive cerebral œdema; no meningitis. On both sides of the transfixed longitudinal sinus a moderately large thrombus was found. The transverse sinus contained fluid blood.

The removal of large pieces of the skull and of the dura, as becomes necessary in cases of large intracranial tumors, is unfortunately too frequently and too easily followed by the rapid development of a fatal cerebral œdema. The transudation which furnishes the cerebro-spinal fluid is facilitated and increased in two ways. First, by increased tension in the arterial vessels, and second, through stasis in the venous sinuses. Arterial tension is the force which causes the formation of the cerebro-spinal fluid; consequently the greater the pressure the larger the quantity of cerebro-spinal fluid. But the very pressure which causes the formation of this fluid also serves to remove it. By an increase of arterial tension, the more rapidly formed fluid must also be removed more rapidly.

Consequently it can never occasion an œdema; as transudation is favored, reabsorption is increased. Venous stasis, however, acts quite differently. Here also dilatation of the capillaries is much more potent than the increase of arterial pressure in separating the cerebro-spinal fluid, *i.e.*, in increasing its volume. The obstructions in the venous flow interfere with the lymph-circulation, causing a slowing and permitting a collection of the fluid in the tissues. But when there is a combination of increased production and diminished flow, œdema will result. The removal of portions of the bony inclosures will cause hyperæmia in the part of the brain exposed as a result of the removal of regular and accustomed counterpressure. The blood-vessels which are most apt to become dilated upon removal of their supports are the veins. The immediate effect, therefore, of removal of a part of the roof of the skull is a venous hyperæmia of the exposed portion of the brain; and it is this venous hyperæmia which afterward leads to œdema of the brain. An old and oft-repeated experiment, which was already performed by Magendie, demonstrates how rapidly and abundantly fluid is replaced even in that variety of venous hyperæmia which ensues after escape of the cerebro-spinal fluid. The fluid is allowed to escape through an opening in the lig. obturator-atlantis; this opening is then closed with the finger and after a little while is again opened; then a stream of newly-formed liquor cerebro spinalis will escape. The high intracranial pressure is a very potent factor in causing reabsorption of the fluid; if it be diminished, as, for instance, by removal of the whole parietal bone, the absorption of the liquor cerebro-spinalis is interfered with and the development and spread of cerebral œdema is undoubtedly favored.

I think this conclusion should not be ignored by those who still advocated the use of the trephine in subcutaneous injuries to the skull, so as to liberate the bruised portion of the brain. This operation will cause an immediate injury to the portion of brain beneath the field of operation, for the liquor cerebro-spinalis will flow to this spot from all sides and will fill the bruised tissue most abundantly; this is shown by the movements of the brain toward the side of least resistance—in this case, the trephined opening. Donders gave a convincing demonstration of this by observing the movements of the lig. occipito-atlanticum postic. before and after trephining the

roof of the cranium. Before the trephining they were quite plain, but after the operation they had disappeared and were only visible at the trephined opening. By replacing the piece of bone which had been removed by the trephine, the movements of the ligament immediately manifested themselves again.

Hemorrhage, which is difficult to control, and the development of an extensive œdema, are the results of extirpating large tumors of the brain. These are disturbances which will seldom permit such an atttack being successful. The danger from œdema is increased through a diminution of arterial pressure resulting from loss of blood during the operation and from obstruction to or at least interference with the venous circulation which the extirpation of one of the larger sinuses must produce. To this must be added the disturbance occasioned in the distribution and relationship of the fluid portions of the cranial contents due to the tumor. With the larger intracranial tumors there is always an increase of the liquor cerebro-spinalis, especially in the ventricles of the brain, a disturbance which is indicated by the occurrence of typical pressure symptoms, especially choked disc. The way in which pressure upon the vena magna Galeni causes hydrocephalus internus is well known. If other factors resulting in the increased formation and the diminished escape of liquor cerebrospinalis, such as do undoubtedly occur after the operation, are added in these cases of extensive overproduction of this fluid, it will be understood how easily an œdema of the brain will become developed and how readily an existing œdema will become increased in amount and extended in area.

If the tumor of the brain has already produced the most serious cerebral disturbances, such as loss of consciousness, sopor, and coma, we will be almost certain to find extensive œedma of the brain at the autopsy. Therefore I regard the attempt to remove tumors of the brain during the comatose state as entirely hopeless. Since the œdema immediately becomes worse as a result of the operation, it will have increased to such an extent in from six to twelve hours as to be incompatible with a continuation of life. I have, however, been called upon more than once to operate upon patients in this stage; I have had to refuse because I considered it too late.

The surgeon who wishes to be careful and critical in the

selection of his cases must not only obtain the typical general manifestations of an endocranial tumor, but the diagnosis must include the seat. The area symptoms of the motor region or of the occipital and temporal lobes must be unquestionably real and direct and not indirect as expressed by Wernicke; they must be produced by the general pressure effects of the tumor. If these become manifest besides the general pressure symptoms, the surgeon then only lacks the knowledge of the limits and size of the growth. Concerning these two points it will only be exceptionally that we will be able to form a diagnosis prior to the exposure of the tumor. Therefore, if the existence and seat of the tumor be certain, I believe it to be proper for the surgeon, acting under the limitations expressed above, to use the chisel and knife in making an exploratory trephining and even in making an exploratory incision into the white substance of the brain. But the operator must be prepared to stop if he finds the tumor too large or diffusely infiltrated; in such a case he must allow the operation originally undertaken only for exploratory purposes to remain uncompleted. Under these conditions, I regard the removal of favorably situated tumors as justifiable and, under especially favorable circumstances, as a duty. It is well known how often we must retreat in the midst of our work, even in a nicely and positively diagnosticated case; for modern surgery has had but very limited experience in this department.

The first two cases, in which larger, endocranial tumors were removed by operation, were instances in which portions of the growth appeared outside the cranium; they were the cases of Macewen and Durante. Macewen's case has probably only become generally known through his address at last year's meeting of the British Medical Association in August. I give a verbatim report:

"In 1879 an idiopathic case came under observation, in which the totality of the symptoms indicated a lesion in the left frontal lobe of the brain. It occurred in a patient the subject of a small tumor above the left eyeball in the orbital cavity. A tumor had previously been removed by me from that position, and it had now recurred. Other symptoms had, however, meanwhile presented themselves. The left pupil was in a state of stabile myosis; there was obscuration of the intelligence, slowness of comprehension, want of mental vigor,

and pain in the head. These pointed to the probability of a lesion in the left frontal lobe, but were not sufficient to permit a diagnosis to be made. The patient was therefore placed under the observation of an educated, skilled nurse. Some weeks later, a series of convulsions occurred, the initial stages of which were carefully recorded by the nurse, without which the key to the brain lesion, as indicated by the convulsions, would have been lost, as, when seen by me, they had become general, and threatened speedy dissolution. The convulsions were at the outset strictly confined to the right side, commencing in the face and arm, and confined to these two parts during the initial attacks; the leg on the same side was affected during the third seizure, and ultimately the convulsions became general with complete loss of consciousness. These phenomena were construed as indicating extension of the irritation to the lower and middle portions of the ascending convolutions; and when this was considered with the former evidence, it was concluded that an irritative lesion existed in the left frontal lobe. On these grounds it was resolved to trephine midway between the centre of the ascending convolutions and the anterior aspect of the cranium. At this point a minute nodule of the size of a barley grain was detected on the outside of the cranium. A large trephine was applied, a disc of bone removed, and a tumor of the dura mater, which was exercising pressure upon the brain, was exposed. It was half an inch in thickness at this point, gradually becoming much thinner and spreading all over the front of the frontal lobe. The tumor was, after a prolonged operation, carefully dissected out, along with the brain membranes, where they were involved in the neoplasm. The patient rapidly recovered, was restored to perfect health, and subsequently was able to gain her own livelihood. She lived for eight years afterward, ultimately becoming affected with chronic Bright's disease, from which she died. The skull and brain were examined, and there was no trace of further tumor growth."

The case is an anomaly. I, at least, have never heard of another case of periosteal sarcoma, for this must be considered as the nature of the primary growth in the orbit, which produced metastases in two different organs (bone and brain) and then was not disseminated further.

Durante,[69] of Rome, claimed priority in extirpating tu-

mors of the brain as opposed to Godlee and Ferrier. In his case, the presence of an intracranial growth was disclosed by the turning of the left eye. The tumor had grown into the orbit behind the globe, through the sphenoidal fissure. The patient, who was affected with melancholia, lost the sense of smell. Removal of a large piece of bone from the frontal down to the upper margin of the orbit. The tumor which occupied the anterior fossa of the left side extended beyond the middle line and had destroyed the lamina cribrosa and had pushed downward the roof of the orbit. It extended backward to the sella turcica. The left frontal lobe was in a condition of marked atrophy. Little hemorrhage during the extirpation. Besides the introduction of a drainage-tube, the wound was packed with antiseptic gauze for ten days. Immediately afterward, the previous symptoms having continued: prominence of the eye, anosmia, deficent memory, uncertainty in movements, tendency to depression and quietness. Three months later, these mental disturbances had improved considerably and were disappearing.

In a third case—mentioned by Birdsall [70] and operated by Weir—operative interference was prompted by the fact that after removal of a sarcoma of the region of the neck, there had been manifestations of the presence of a tumor of the brain. The case will serve here as a discouraging example.

It is to be hoped that this will be the last and only imitation of Macewen's lucky operation in a case of multiple metastatic sarcoma of the brain.

A woman of twenty-six had been operated upon four times on account of sarcoma of the neck. During the last operation, the brachial plexus had become injured and a paralysis of the left arm had resulted. Involuntary micturition and defecation set in. After this, spasms in the left leg followed by paralysis of this limb. Then spasms and numbness of the right hand. During the last three weeks intense headaches. August 11th, 1887, the tumor which was believed to be in the upper portion of the fissure of Rolando, was sought for. Dura, and after incision into the latter also the substance of the brain, bulged into the wound. Neither the finger nor the exploratory needle could demonstrate the existence of a tumor either on the surface nor in the depths of the convolutions. A part of the brain substance which had bulged forward was

cut off, and the rather profuse hemorrhage was controlled by the thermo-cautery. One week later, hernia cerebri developed of the size of a hen's egg. No reaction, but death after two months, with increasing symptoms of an affection of the spinal cord. At the autopsy, no tumor was found in the cerebrum, but there was a spindle-celled sarcoma originating in the left cerebellar hemisphere and pushing the medulla toward the right and forward.

The history of the case is too brief for me to form an opinion regarding the circumstances which rendered it possible to mistake a paralysis produced by the spinal cord for one emanating from the motor area of the cortex of the brain. My criticism will be expressed sufficiently in wondering how an operation could have been thought of in a case of metastatic sarcoma, of which there were undoubted indications.

In the three cases just mentioned, there were, in addition to the brain symptoms pointing to the presence of an intracranial growth, other and quite definite indications of this condition: the signs of a retrobulbar tumor extending into the orbit from above in Durante's case, the pre-existence of an orbital sarcoma and the presence of a small sarcoma of the bone in the situation at which trephining was done, in Macewen's case, and finally the recurrence of sarcoma of the neck three times, after which there was the formation of metastases in the interior of the brain, in the case belonging to Birdsall.

November 25th, 1884, Bennett and Godlee first ventured to open the skull in a case which had been diagnosticated as a tumor.[71] The case attracted even more attention than it deserved, because it was reported at the time when the patient had recovered from the immediate effects of the procedure and was perfectly well. Unfortunately he died four weeks after the operation, not as a direct result of the latter, but from infection of the wound.[72]

The case occurred in a man of twenty-five, who during the preceding four years had been subject to attacks of severe headache, especially in the parietal region, with almost uncontrollable vomiting and retching. The general condition and intelligence were not affected, but there was an optic neuritis on both sides. Speech, hearing, sensibility, and ocular motility were normal. On the other hand, there was complete paralysis of the left hand and fingers, inability to pronate and

to supinate the left forearm, diminished mobility of the left shoulder-joint, and a very slight paresis of the left leg and left side of the face. At times there were clonic spasms of the paralyzed muscles. The tendon-reflexes on the left side were more marked than those on the right. There was distinct tenderness of the right parietal region near the sagittal suture. Bennett diagnosticated a tumor situated on the right side in the neighborhood of the upper third of the fissure of Rolando; and Godlee, after determining the corresponding area on the surface of the brain, applied the trephine, making an opening of $2 \times 1\frac{3}{4}$ inches. At the bottom of this opening, after the dura was removed, a peculiar pale-yellow cerebral convolution presented itself. An incision led to a firm, lobed, solid, and well-circumscribed tumor, which could easily be removed with a spatula. It was found to be a glioma, conical in shape, about the size of a walnut. The hemorrhage, which was considerable, was controlled with the galvano-cautery, and the cavity, which was $1\frac{1}{2}$ inches deep, was drained. Then the dura and integument were closed by stitches and an antiseptic dressing applied. Patient stood the operation very well. His intelligence remained intact and his disposition lively. There was no pain. There was no change in the paralyses.

In changing the dresings daily, swelling of the margins of the wound and suppuration from the depths were found on the fourth day. The skin of the face and scalp were œdematous. Soon a portion of the surface of the brain about half the size of an orange bulged through the opening. In the eighth day this prolapse was cut off. Although the patient's general condition remained good immediately after, three weeks later a purulent meningitis set in, as a result of which the patient died December 23d. The defect in the right cerebral hemisphere took in almost the entire posterior central convolution, only the upper and lower portions of this being preserved. Hernia cerebri. Purulent meningitis, most marked at the base.

It is certain that this case was an uncommonly favorable one; it is equally certain that there must have been some error in the after-treatment, for otherwise antisepsis must have guarded against the occurrence of prolapse and of purulent meningitis. I do not hesitate to say that the iodoform tampon recommended by me would have controlled the hemorrhage

more effectually than the galvano-cautery did, and at the same time, by exerting pressure, would have prevented the prolapse.

The following is the case of Hirschfelder:[73] A laborer of thirty-seven was subject, in the course of a year, to headaches, epileptiform attacks, vertigo, amaurosis, loss of motion and sensibility in the left arm, occasional spasmodic contractions of the latter and of the left side of the face, and a similar paralysis of the left leg. Tendon reflexes exaggerated on both sides, but especially on the left side. Since the face, arm, and leg of the left side were involved, a tumor in the neighborhood of the right fissure of Rolando was diagnosed. Although three trephinings resulted in an opening three inches in diameter, the operator was not able to remove the diffuse and extensive, very soft growth which permeated the brain for quite a distance. It was necessary therefore to be content with the removal of only 2½ cm. of the new growth which proved to be a glioma. During the following eight days which preceded death, there was a considerable discharge of blood, fetid material, and pus, in which glioma elements were found. No autopsy was permitted.

Horsley was the first successful operator. In 1887 he presented, in the form of tables, the cases in which he had opened the skull, partly on account of brain tumor and partly on account of epilepsy.[74] Among these ten cases, two refer to operations upon endocranial tumors, besides the two cases of tuberculosis of the brain which have already been referred to above. The immediate result in both cases was favorable, but one of them died soon after, from recurrence. I can only state, regarding these cases, that which is contained in the table, for a detailed report has not yet appeared.

A man of thirty eight had been suffering from paralysis of the left side and from epileptiform convulsions which started in the left shoulder; after being in a semi-comatose condition for ten days, he was operated upon September 23d, 1886. A glioma weighing 4½ ounces was removed from the upper part of the area for movements of the arm in the right hemisphere, through the trephine opening. The incision was carried purely into the depths of the white substance of the brain on all sides of the tumor into healthy tissue. Strict Listerian dressing with carbolized gauze. Drainage-tube was left in for two days. The greater part of the wound healed by primary

union. A small part of the wound was reopened on the eighth day so as to allow a light-colored, serous fluid to escape; it was kept open thirty days. Highest temperature on right side, 101.5°; on the paralyzed side, 102.5°. The sensorium became completely free after the operation and remained so for two and a half months. The epileptic attacks did not recur. However, three months after the operation, there was a rapidly increasing recurrence from which patient died, March 18th, 1887.

The second patient of Horsley's was a man of thirty-seven, whose general health was good with the exception of a constant headache. His right hand and forearm were completely paralyzed, his right leg incompletely. The paralysis had developed and increased gradually. Epileptiform convulsions, which began in the fingers of the right hand; these had not, however, occurred since three months. December 7th, 1886, trephining and removal of a tumor weighing 4½ ounces. Strict Listerism with spray. Absolute union of the edges of the wound, except at the opening for the drainage-tube. Highest temperature, 100.4°. Condition of general health and of intelligence remained good. No headaches, no convulsions, no increase in paralyses up to the date of publication of this extraordinarily important case, April, 1887—four months after the operation.

To this very important and interesting case of Horsley's may be added two cases of last year, which equal it in favorable result. The one comes next to the case of Horsley's in regard to size of the growth which was removed; it belonged to Keen. The other case was diagnosed by Seguin and operated upon by Weir.

Keen[75] removed a large fibroma with the very best result: A carriage-maker, aged twenty-six, married three years, had one healthy child; when three years old, fell from the window a distance of several feet, faling upon the head; following this there was swelling of the frontal region and patient lay in a comatose condition for some time. During his fifth year, after an attack of measles, he developed an otorrhœa of the right side, which recurred at variable intervals and caused considerable damage to hearing. In August, 1886, left ear also became partially deaf. During boyhood complained considerably of frontal headaches. In the autumn of 1884 he began to suffer from neuralgic pains on the left side of the head, extending

from the frontal eminence to the occiput; these pains were increased upon pressure over the left parietal, and here a small, white cicatrix was found. February, 1885, he was seized with violent epileptic attacks followed by severe headache lasting several days. The seizures occurred once or twice during the week. The headaches gradually increased in severity and duration. By the end of April the right arm became paralyzed, then the right leg, and finally the right side of the face, the paralysis progressing gradually. There was more interference with motion than with sensibility, which was, however, also affected. Right pupil widely dilated and does not respond to light. Vision of right eye was imperfect. There was aphasia; he made marked efforts to converse, but could not. Pulse 60; respirations 16; obstinate constipation; insomnia; no fever. This was the condition of the patient June, 1885, when Keen first saw him. Iodide of potassium in large doses, also arsenic and laxatives were prescribed.

The pain began to diminish and at the end of three weeks he was nearly free. Paralysis began to improve in the arm and leg. Finally, the aphasia began to disappear; he would write out any questions he desired to ask. His eyesight began to fail and August, 1885, he became totally blind, first in the right eye and later in the left. At the end of two months his left eye gradually improved so that he could again walk in the streets. The right eye remained blind for several months, when suddenly the sight returned in it, remained for a few hours and left as quickly as it came. This phenomenon has since occurred frequently, not only in the right eye but also in left. The epileptic attacks continued with diminished violence and frequency until November, 1886, since which time he has been exempt. Mental condition in general, weak; memory good. His condition May 30th, 1887, was as follows. Headaches moderately severe, generally lasting from half an hour to several hours and occurring from two or three times a week to one in several weeks. Speech slow and hesitating. During the seizures, eyes turn to the right and face turns in same direction; patient retains consciousness during the attacks; he feels as though smothered; then unconsciousness follows. The right hand is clenched over closed thumb. These alternate with minor attacks. No paralysis of legs visible in walking; power of hand about equal on the two sides. The

face is no longer paralyzed. Right pupil wider than left. Right eye deviates slightly upward and outward. The left side of skull is now no longer tender. Careful examination reveals a diminution in the sight of the right eye; patient can scarcely count fingers at six inches.

Left-sided homonymous hemianopsia. Opacities in vitreous and optic nerve atrophy on both sides. Subsequently the epileptic seizures became more severe and frontal headaches and vomiting recurred.

In the fall of 1887 the patient was examined by Weir Mitchell, Morris Lewis, and George Harlan, and an operation in December agreed upon. The scar finally settled upon as that resulting from his accident at three years of age "was a quarter of an inch long, and half an inch in front and above the superior stephanion, two and a quarter inches to the left of the middle line, and three inches behind the external angular process. It was now tender both to pressure and to a slight blow. The temperature over the scar on this side of the head was 95.5° F., and in a corresponding position on the right side 94.4° F. Dynamometer, right hand 30°, and left 35°. Knee-jerk, left normal; right subnormal. Tactile sensibility in both hands normal." December 15th, operation beginning with an incision through the scar; no scar was found on the bone when uncovered. Then a flap was cut and a piece of bone, 1½ inches in diameter, was removed; bone thin, dura thickened; beneath it a hard growth extending in all directions beyond the trephine opening. A second button was then removed posterior to the first, and the intervening bone was broken away so that an opening 2½×3 inches was produced. The upper margin reached to within ¾ inch of the middle line, when the border of the tumor was fully exposed. The lower portion of the tumor dipped behind the squamous portion of the temporal bone. The dura was found to be adherent toward the centre of what corresponded to the scar and hence was severed all around, after which the growth could easily be enucleated with the finger. The tumor weighed three ounces forty-nine grains, and was 7¼ inches in circumference in its greatest axis; it was pinkish-white, nodular on surface, and lobular. It reached backward nearly to the fissure of Rolando, forward (two and a half inches) into the bases of the three frontal convolutions, upward into the external part of

the first frontal convolution, and downward nearly to the fissure of Sylvius. Microscopical examination showed a fibromatous structure. No vessels required ligation in the dura, but several large veins of the brain poured out abundant streams of blood during and after enucleation of the tumor; three or four of these were tied with difficulty owing to the ligatures cutting through. Finally they were ligated, but still the bleeding was rather profuse and the wound was therefore douched with hot water, and direct pressure by sponges was tried, and thus the bleeding was finally controlled. During the time occupied by controlling the hemorrhage, the cavity left by the tumor had been filled up nearly one-half by the resilient brain tissue. Rubber fenestrated drains were introduced at two points in the posterior border of the wound and a bundle of horse-hair was carried from one opening to the other across the wound. The scalp-wound was next united and sponges placed upon the flap so as to depress it. Sublimate dressing. The operation lasted nearly two hours, most of this time being required by trephining and checking the hemorrhage. The patient bore the operation well. When placed in bed he rolled persistently to the left side and drew both legs up. He vomited often, causing blood to appear in the dressing and necessitating a change of dressing twelve hours after the operation, and again several times in the course of the succeeding days. He was perfectly conscious, but aphasia was somewhat marked. Little pain. Temperature did not rise higher than 100.4°. On the third day a large clot weighing about four ounces was removed from beneath the flaps. There was an immediate improvement in the aphasia which had become worse after the operation. On the fourth day, removal of the first drainage-tube: on the seventh, removal of the sutures, since the wound had almost completely united. Æsthesiometer showed no anæsthesia; mental condition clear. During the second week, fever over 102.2°; this subsided at the beginning of the third week. On the eighth day, aphasia increased, lower part of face on right side less movable; required catheterization. On the ninth day, paresis of right arm which increased to complete paralysis on the tenth. Deafness added to the aphasia. On the eleventh day, paralysis of right leg. The wound was reopened and a quantity of discolored, soft tissue escaped. After this the fever diminished, the deafness

and asphasia improved, and the flap applied itself more closely. Hernia cerebri occurred through the crescentic margins of the wound. During the third week, while there was still an elevation of temperature now and then, there was an improvement in the power of the right leg and later this change also affected the right side of the face. The prolapse of brain, covered with granulations, increased and, beside pus, also gave rise to a large quantity of clear fluid; this did not cease until the end of the fifth week. On the twenty-seventh day, patient began to move the right arm. On the fifth, fever recurred and paralyses increased; when fever subsided these also improved. By the fortieth day, the movements of the arm and of the fingers had improved so that he used them once, but could not repeat this. At the wrist he had flexion but no extension. After this there was a steady gain in extent and power of motions in all directions.

The hernia was covered with granulations, but as they showed very little tendency to cicatrize on the thirty-fourth to the fiftieth day, thirty-four skin-grafts from his arm were used to hasten the process; of these, all but four adhered. On the seventy-first day cicatrization was complete. On the seventy-fifth day, the site of the hernia, instead of being an elevation, had suddenly changed to a deep hollow. The patient went home well on the eighty-fourth day. Dynamometer still shows decrease in power. April 19th, patient had an epileptic attack.

Keen says: "I confess that I was very reluctantly brought to the conclusion not to operate when this patient first came to me. When first seen, the site of the injury was extremely doubtful. Shaving the head disclosed scars, and the clinical history reported no scar of sufficient moment to be remembered. Even which side of the head had been injured was doubtful, and the most contradictory statements were made as to the initial symptoms of his fits. The old suppuration of the right ear raised great doubts as to how far that might be the cause of a right-sided cerebral mischief, while, still further to complicate the problem, the examining ophthalmologist was of opinion that the ocular symptoms pointed to an old syphilitic lesion, in spite of the denial of the patient."

The absence of any improvement during the first months, when paralysis, blindness, and aphasia were present, was in

contrast to the subsequent changes when the paralyses disappeared, and excepting the ear and eye symptoms, the difficulty in speech and the epilepsy, all functions remained normal.

Weir and Seguin report:[76] A German brewer, thirty-nine year old, robust, in good health until the fall of 1882; then he became ill with malaria accompanied by a good deal of headache. One day, when he did not feel well, his wife observed spasms of the right cheek and a turning of the head to the right side; there was no loss of consciousness. One year later the attack was repeated and during the third year he had an occasional attack in the night. He was well otherwise, with the exception of an occasional headache, when one day he fell unconscious and bit his tongue; he has had similar attacks at long intervals since; they lasted only a few seconds, but left him very weak. The loss of consciousness was preceded by twitching of the right hand and arm and of the right side of the face. The attacks became fewer after taking bromide of potassium. No ataxia. Patellar reflexes normal. In August, 1886, Seguin noted that patient could speak individual words, such as "water," "ice," but could not talk; speech much affected. During the last eight or nine months, the right upper extremity had become weaker and "numb." Memory much impaired. Pupils even. Optic nerve entrance normal, also ocular motility. The muscles of lower part of right side of face are paretic; tongue deviates slightly toward the right in protrusion. Right eye is not closed completely. Gait normal. Power equal in legs, less in right arm than left. Sensibility in general unchanged except a slight diminution over the right cheek, demonstrated by the use of the æsthesiometer. Last epileptic attacks June 10th and August 11th, 16th, and 18th. August 21st, 23d, and 25th, had an intermittent fever, with chills, etc. At that time the diagnosis of a tumor of the left motor zone in the facial centre was made. In September, speech had become worse and this was noticed by the patient. During an attack of fever, September 13th, he had severe left-sided headache, feeling of numbness and weight in entire right upper extremity; not present elsewhere. Complained of drowsiness in daytime. Slight tactile anæsthesia on thumb and index finger of right hand. Muscular sense, when eyes are closed, is normal on both sides.

In October, there were several epileptic seizures, following twitchings of right side of face, with this side of the face redder and warmer than the other. Has had more severe and more constant pain in the left parietal region. Facial hemi-paresis is much worse. The anæsthesia, though very slight, is demonstrable on right face and hand. Percussion develops tenderness over an oval area $2\frac{1}{2} \times 2$ inches, above the left ear, and overlying the motor zone. Measurements with the surface thermometer do not show any essential difference between right and left. November 17th, Weir trephined under the usual antiseptic precautions, the middle of the button corresponding to the lower half of the left anterior central convolution, $1\frac{1}{2}$ inches above and somewhat in front of the auricle. A second button was removed just in front of the first, and a little above it. The intervening portions of bone were gnawed away with a double gouge forceps, and the cranial opening enlarged on all sides in the same way until it reached an area of 3×2 inches. The dura presented a normal appearance and pulsated freely; it was cut into for about three-quarters of the circumference of the opening, thus forming a flap which was turned up. The blood-vessels over and under the dura were tied without difficulty. Nothing abnormal was seen on the exposed surface of the brain; the finger recognized no tumor or abnormality, but when firm pressure was used, a deep resistance of a hard mass of small size underneath a previously suspected convolution situated most posteriorly and having a somewhat violet color. The convolution was gently parted with the finger-nail and a director: and at a depth of nearly an inch a mass was exposed to the touch and subsequently indefinitely to sight by means of gently used retractors, made of bent spoon handles. It was about the size of an almond, not encapsulated, and seemingly infiltrated into the brain tissue. It was removed with a Volkmann's spoon which had been previously blunted for the purpose. After the tumor had been taken away, a separate hard piece about the size of a pea was recognized and removed. The cavity formed seemed to have normal walls. A rubber drainage-tube was carried to the bottom of the cavity and out through the posterior margin of the wound. Disinfection with 1:5,000 sublimate solution. The dura was stitched together except over a small area where the tube emerged, and the discs of bone

and a number of bone fragments were replaced over it; these had been kept in a towel wet with 1 to 60 carbolic acid, and kept at a suitable temperature by immersing the jar containing them in warm water for over an hour. Iodoform gauze was placed in the lower part of the wound; the rest was sutured; sublimate dressing. The tumor lay entirely within the white substance of the brain and microscopical examination showed it to be a round-celled sarcoma. The subsequent course was, under appropriate treatment, a favorable one. The use of large, increasing doses of bromide of potassium was continued. Immediately after the operation there was a temporary, complete paralysis of the right extremities, and complete aphasia. Three weeks after the operation, examination showed patient in bed, perfectly well, without convulsions. There was an improvement in the paresis of the lower branches of the facial which had existed before the operation. Tongue could be protruded straight; pupils and optic nerve normal. The weakness of the right hand is probably less marked, but the emaciation of its small muscles is prominent. Light touch with the finger is felt equally on both sides of the face, except the lip which is more sensitive on the left side; this is also proven by the æsthesiometer. Sensibility over right hand and forearm good, though slightly blunted. Voluntary movements of right leg free and good. December 10th, had an attack commencing with twitching of the right side of face and right forearm, followed by loss of consciousness and finally general convulsion; short subsequent coma.

January 23d. Has had no marked attack in two weeks; only an occasional twitching of the facial muscles. Has also had a few vertiginous or faint feelings. There is still some paresis of lower branches of facial of right side. Right upper extremity is paretic, lower normal. Only slight difference in sensibility on the two sides. End of thumb and fingers still show plainly the loss of sensibility.

March 8th. There is much more aphasia than at any time; so much as to render tests of sensibility unreliable. On 19th and 25th, convulsive attacks limited to the right hand. April 3d, improved, but lower part of right side of face less expressive than left. Otherwise little change.

It is obvious that there were no signs of a return of the sarcoma five months after its removal; nor could any be dis-

covered at the last visit of the patient in June. The bromides of sodium and potassium were continued uninterruptedly.

During the summer of 1888, patient went to Germany and, bearing a card from Seguin, visited Prof. Bernhardt in Berlin; the latter kindly gave me some notes concerning the patient. During the voyage he had had one epileptic seizure; he can move the right extremities pretty well; mouth is drawn to the left on account of paralysis of the lower branches of the facial; speech is still difficult and sometimes he is at a loss for a word or he cannot find the right word at once. He does not write, since his right hand cannot hold the pen.

The six cases just mentioned, of Bennett-Godlee, Hirschfelder, Horsley, Keen, and Weir-Seguin, to which also belongs a seventh, that of Birdsall spoken of still further above, are cases in which an endocranial tumor was diagnosticated and found, at the operation, from a consideration of brain symptoms only. Two of these seven cases (Birdsall and Hirschfelder) terminated fatally as a result of the operation. In the third case (Bennett-Godlee) death resulted at the end of the first week as a result of wound infection. Consequently four cases recovered. Of these, one died in three months as a result of residual growth (Horsley). Hence three of those operated upon remained well; a result the importance of which cannot be denied.

I have endeavored to determine these relations since nowadays—at least among the neuropathologists of Germany—there is a tendency to exaggerate the favorable results of the extirpation of tumors of the brain. On this account I must also consider those cases in which the operation was done under a mistaken diagnosis, not without injury to the patient, those cases in which the tumor was not found, and finally those in which the operation had to be abandoned in an unfinished condition, on account of too great size, or too extensive adhesions of the growth.

To the cases of the last category belongs one of Heath:[77] A man twenty years old, given to drinking, fell upon his head in his sixth year. In 1886, he fell upon the right temporal region, followed by a loss of sight in the right eye the next day. Before this, since 1883, he had epileptic seizures in which head, arm, and shoulder were drawn toward the left side. Constant headache. Limited intelligence. Optic atrophy on right side;

later also beginning on left side. Anosmia on right side. No anæsthesia. Sensitive point on percussion, 1¼ cm. behind the zygomatic process of the frontal bone, two inches above the zygomatic arch. Trephining, October 13th, 1886, corresponding to the sensitive spot. A deep-seated tumor, adherent to the floor of the anterior fossa of the skull, was found which could not be removed on account of its firm attachments. After thirteen months general condition was good, but headaches and epileptic convulsions had increased.

The tumor was not found in the second case of Birdsall's (mentioned above) in which there was a metastatic sarcoma of the cerebellum which had extended to the spinal cord, though the operator sought for the source of the disturbances, which had been observed, in the region of the central convolutions. To this class also belongs the following report of Hammond's:[78]

The patient, a married woman, complained of a severe pain an inch above the right ear and had left hemiplegia. At the time her symptoms began she was twenty-nine years of age; she had had four children; during the progress of her disease she had a fifth child. All were well and strong. Her sickness lasted about two years and a half. It commenced by a sudden loss of consciousness, and convulsions limited to the left side. The left side of the face became and remained paralyzed. About a year later she noticed gradual loss of power in the left arm; things dropped from the hand. At the end of another year the left arm was completely paralyzed. She then noticed increasing weakness in the left leg. When Dr. Hammond first saw her, she was able to stand and walk slowly. While the paralysis was extending she had four or five epileptic seizures. Confined to the left side, headache developed about the time that the leg became affected, grew more constant, and was described as agonizing.

She denied syphilis. Physical examination showed loss of motion on the left side of the face, tongue, and soft velum, of the left arm, and partial loss of motion in the left leg. There was no disturbance of sensibility of any kind. The reflexes were exaggerated on both sides. Sight, hearing, smell, and taste were normal. Choked disc on both sides was observed at a subsequent examination.

Under treatment, the headaches were controlled to a con-

siderable extent. Dr. Hammond's diagnosis was probable cerebral tumor involving the cortical or subcortical substance of the motor areas. An operation was refused. The patient grew worse until the 20th of March, 1887, when her husband asked that the operation be performed. Dr. Spitzka then saw the patient with him and made two examinations. He coincided in the diagnosis. The operation was performed in the hospital March 30th; the portions of the skull were removed by the electro-osteotome. Four buttons were removed by the electro-trephine. These were connected by straight lines made with the circular saw. The diameter of the opening was over two inches. A crucial incision was made through the dura. A hypodermatic needle was introduced in different directions, but no fluid was withdrawn. The dura was closed, a drainage-tube was introduced, the skin flaps were sewed up. The patient was put to bed. She lived only twenty-one hours, remaining unconscious after the operation. Prior to the operation she had become completely paralyzed in the left lower limb; she had delusions and hallucinations; she talked incoherently; bedsores developed.

The autopsy showed little hernial protrusion in the wound; the cortical substance here was thin. Incision through the motor region revealed three cysts in a line deep in the white substance. The fluid in the cysts had been only partially examined; it was serum, and contained broken-down brain substance. The cysts were close together, and in a position to affect the face, arm, and leg centres.

The serous contents of the cysts were not examined more fully, or perhaps the new formation might have turned out to be hydatids.

The diagnosis in the previous case was exactly correct; but the cysts were situated beneath and not within the cortex; and since they did not feel harder than the rest of the brain, they were not detected by the finger of the operator.

Last year I operated under a mistaken diagnosis:

The case occurred in an officer, twenty-five years old, coming from a healthy family; he had measles when a child; two years ago he had had gonorrhœa and soon after was in the military hospital at Mühlhausen for three months, with an ulcer of the right leg which he attributed to tight boots. Sometimes he had headaches which he thought were due to exer-

cising for a long time with his helmet on. On questioning, he states that he has a small scar in the middle of the left parietal region, produced through the falling of a knife whilst he was employed in a butcher establishment six or seven years previously.

Beginning of April he came to Berlin to enter a local regiment. Soon after, he began to suffer from violent headaches. April 19th, he says, he noticed a weakness in the right arm and the next morning he stumbled on the stairs and in this way first noticed a weakness in the right leg. During the same afternoon he lay upon a sofa and fell off suddenly without being aware how this had happened; he could get up and go into a basement where he again felt all right. However, one hour after, he fell from a chair and lost consciousness. This soon returned, but he could no longer speak, and at the same time his right side, arm, and leg had become paralyzed. No satisfactory answers were given in reply to the question at what time the rigidity of limbs observed upon admission had first been noticed. His father said they had been entirely limp at first. Patient was confined to bed for five weeks and treated with large doses of iodide of potassium. This was followed by an improvement in his condition; he could walk again and raise the arm somewhat from the shoulder; he was also able to say a few words. This improvement continued during the summer when he was under electrical treatment. October 1st, 1887, he again suddenly fell from a chair and then lost consciousness for a short time. Since this attack he often had twitchings in the paralyzed limbs. Besides these he also had epileptic attacks at night on three occasions. These began with a cry during sleep and violent twitching in the paralyzed right arm, soon spreading over the whole body. He was not conscious of these attacks. He frothed at the mouth and bit his tongue. He had had his last attack of this sort January 2d, 1888. Upon admission to the clinic, January 10th, he looked robust and healthy, of strong constitution, well developed muscles, healthy respiratory and digestive organs. Appetite and digestion excellent. No fever. No headaches. He experiences pain when a spot about 3 cm. square above the right auricle is percussed with the hammer or finger. While the face is in repose scarcely any difference can be seen between the right and left sides; but when he tries to move the parts of the face

the paralysis of the lower branches of the facial becomes apparent, while the eye of the corresponding side can be closed and the skin can be wrinkled. The tongue deviates slightly toward the right, uvula hangs straight, the pillars of the fauces are symmetrical. The right arm hangs from the shoulder, slightly bent at the elbow. The hand is cyanotic and cold; fingers flexed. All muscles of the arm, including the pectoralis major, are in a condition of moderate tension. It takes but little force, however, to extend the flexed fingers and to abduct the arm from the shoulder; but patient complains of pain when the latter motion is executed. He, himself, can scarcely raise the arm to 40°, during which there is a continuance of the flexion at the elbow-joint. He does not succeed in moving fingers actively nor in extending or continuing the flexion of the forearm at the elbow-joint. In walking, he sways the right leg in a half-circle externally and anteriorly, before he places it upon the ground. He cannot move his toes. The movements at the hip-joint are the most perfect of any of the lower extremity, next the knee-joint, and still less perfectly the ankle-joint. Passive movements are free and painless. In moving the left arm there is simultaneous motion in the right; the same is the case, though less constantly, when the right leg is moved. Tendon reflexes are stronger on the right than on the left side. Right, ankle-clonus; left, none. When movements of the right arm and leg are attempted, these muscles begin to be the seat of tremor which occurs in attacks, is severe for a time and then diminishes. This tremor and these twitchings are especially marked in the quadriceps muscle, when patient stands.

Tactile sensibility is diminished upon the right side of the face. The right side of neck and right shoulder are insensitive to the prick of a needle. Tactile sensibility in right arm is markedly diminished, less so in right leg, as measured with the two points of a compass. In the same way the sensibility to weight is diminished on the right side, so that patient does not feel the weight of eight pounds. With eyes closed, patient can tell the position in which the right arm is placed, but not that of the right hand and fingers. Vesical and rectal functions normal. Heart normal.

No abnormality of fundus oculi; no reduction in field. Intelligence preserved. Patient understands the examinations

and himself calls attention to the differences between right and left sides. He answers all questions as far as his power of speech permits. He recognizes all objects, but cannot name them. He can repeat short sentences with difficulty. Certain words such as "well," "yes," "no," he employs frequently and correctly. He can write his name, and sentences which he has written frequently, with the left hand. Occasionally he cannot write at all or puts down the wrong words instead of those dictated to him.

A clover-shaped, white scar with brown margins was found upon the right leg: on the glans penis a pigmented spot, about the size of a five-cent piece was seen; but no scar. These two changes served as guides to the diagnosis. On account of its form, I regarded the scar on the leg as produced by a syphilide. In consequence, patient was put upon inunctions to the point of salivation and this was followed by the administration of large doses of iodide of potassium; but there was no result. Several times each week, violent twitchings in the paralyzed lower extremity were observed; these lasted from two to six minutes, and were not accompanied by any loss of consciousness. He also had one real epileptic attack at night and one in the morning.

I examined the patient frequently with Profs. Mendel and Jastrowitz, in the course of the three months during which I had him under observation, his condition remaining the same except the increasing frequency and severity of the tremors and twitchings. We did not arrive at a definite diagnosis. After considerable discussion with Prof. Jastrowitz, we concluded to make a large opening into the skull so as to ascertain whether the extensive affection of the left hemisphere, which we assumed existed, consisted of the formation of a tumor, and if such were found, whether it extended to the cortex or could be reached immediately beneath the cortex. If we did not find any new growth, which we concluded from the lengthy duration must be a benign one, or possibly an obstinate syphiloma, we were to stop here and to close the wound.

I will recapitulate briefly the points which led us to think of a tumor in the cortex of the brain: the paralysis and spasms in the paralyzed extremities, the early contractures of the muscles of the right arm, which probably occurred soon after the paralysis, the lasting aphasia, the loss of muscular sense

in the hand and forearm, the epileptic seizures, and, finally, also the absence of any affection of the heart.

It is evident what was lacking for this diagnosis: in the first place, there was an absence of persistent headache and of optic neuritis; then, the sudden apoplectiform commencement did not correspond with the usual history of development of a tumor of the brain. The regular tenderness upon percussion over the portion of the left parietal bone already described seemed important to me, as pointing to implication of the cortex; this tenderness was so great that patient shrank from every stroke of the finger.

On the 21st of April I chiselled away a piece of the skull, $7 \times 7\frac{1}{2}$ cm., after making a large skin flap with the base upward. The situation of this piece was such that from an estimation of the direction of the fissure of Sylvius and the fissure of Rolando, the motor region would be exposed, with the exception of its upper third so as not to get too close to the longitudinal sinus. The dura which appeared in the wound pulsated plainly only at its anterior portion; but not in its posterior part. In separating it, the posterior portion was found specially adherent to the deeper parts. In the greater part of the posterior portion of the wound a yellowish, even surface could be plainly seen, while in front, one or two convolutions appeared through the dull pia mater. An incision into the regular and even-looking stratum extended through a layer about 2 mm. thick, consisting of firm, œdematous brain tissue, and then there was a discharge of a quantity of light yellow fluid in which some flakes and threads floated. The surface of the brain at once became depressed deeply. The cavity thus formed was about the size of a hen's egg. Its walls presented large-meshed, soft connective tissue, of brown color here and there; strings and broader pieces hung into this cavity from its walls, resembling a torn net. Consequently we had here a cavity filled with fluid and permeated and surrounded by soft, loose, connective tissue, the whole representing the remains of an apoplectic mass. The cavity extended far into the white substance. Hemorrhage from the cavity was slight, but that from the blood-vessels of the pia was more troublesome; previously a large branch of the anterior branch of the meningeal artery had been tied after cutting through the dura. The cavity was loosely packed with iodo-

form gauze and the skin flaps were only partly replaced; upon the iodoform gauze which appeared through the widely-gapping wound, our usual dressing with sterilized gauze and sterilized bandages was applied.

The patient seemed to have stood the operation well. Pulse 60, no pain. In the evening the outer layers of the dressing, which had become soaked with fluid tinged with blood, were changed.

On the 23d, the iodoform tampon was removed, patient being under the influence of an anæsthetic. As there continued to be a very abundant discharge of fluid resembling liquor cerebro-spinalis, it seemed probable to me that the cystic degenerated connective tissue extended into the lateral ventricle. This induced me to introduce a drainage-tube and to close the wound in the integument around it. During the anæsthesia, there were severe convulsive tremors of the right arm and leg. The condition of paralysis and contracture was worse after the operation and the aphasia was complete.

During the night of the 24th and 25th, he had two severe epileptic seizures followed by a deep sleep, lasting many hours.

On the 26th, patient could again talk and used more words than at any time during his stay in the clinic. The drainage-tube was removed.

On the 27th, the skin flaps seem pushed out and this surface pulsates plainly. On this account the recent adhesions were loosened all around. There was an escape of a large quantity of pretty clear fluid. The flaps became depressed. From this time on, oozing of cerebro-spinal fluid continued, so that the bandages, which became soaked, had to be changed several times a day. In the mean while, evening temperature rose from 100.4° to 101.5°.

May 1st, great restlessness, stiffness of the muscles of the neck. On the 5th this stiffness had diminished, as had also the evening elevations of temperature. On the 8th, patient was fully conscious and clear, and answered briefly, with difficulty and slowly, but correctly. The wound had to be reopened repeatedly, since, as soon as the escape of fluid was hindered by adhesions, fever, slowing of the pulse, and rigidity of the neck would set in.

May 18th, again had a severe epileptic seizure, after which there was no return of consciousness. Patient was restless,

turning upon his right side after being placed upon his back. Stiffness of the muscles of the neck. Pulse 116. No response to loud calling. Paralysis of facial more marked than formerly. Eyes turned toward the right. This condition persisted up to the time of his death, May 20th, over four weeks after the operation. The autopsy showed a collection of turbid, brownish fluid which communicated by means of a narrowed portion with the cavity in the white substance of the brain of about the size of a hen's egg; this cavity was filled with similar liquid which, however, was richer in pus, and had a narrow communication with the right lateral ventricle. In the latter and its fellow there was similar turbid fluid.

This case demonstrates how difficult it is to prevent decomposition and purulent degeneration of the cerebro-spinal fluid through the entrance of infection, when this fluid is escaping through a large wound; for the final cause of death was undoubtedly leptomeningitis as shown by the clouding in the contents of the ventricles. It is also a warning to us not to regard the exploratory puncture, or, what is the same in these cases, the exploratory incision, as a matter of no consequence. If, in making this incision, we could always avoid disturbing the brain, I, also, would regard the proceeding as one unaccompanied by any risks to life or at most resulting in a future danger dependent upon thickening of the meninges and adhesions of the surface of the brain to the scar. But it is seldom the case that we can avoid disturbing the brain. We expose the brain in order to remove from it the disturbing factor which we are in search of. For this purpose we must palpate, separate convolutions, cut into them or tear them; and we can never know how far we are led by that which we find. Had I known that the firm mass which I encountered was the wall of a cyst communicating with the lateral ventricle, I certainly would not have opened it. I only incised it in order to see what there was, and in doing this I was taken further than I had anticipated. I believe this will be the end of many an exploratory incision, just as, fortunately, exploratory laparatomies are coming more and more into discredit. With these also, we often intend merely to examine whether the gastric or the intestinal cancer can be removed; but in making the necessary examinations we tear bleeding adhesions or even into the wall of the neighboring intestine, and thus, in-

stead of an "innocent exploratory incision," we are forced to do a great, difficult, and yet incomplete operation.

I have not been able to find additional cases in which tumors of the brain were sought for. The cases which are counted as belonging to this class elsewhere, refer to traumatic changes in the surface of the skull, beneath which changes in the bone, membranes, or the surface of the brain were sought for, the removal of which was to favor the cure of spasms or paralyses. Other cases are repeated and mentioned twice; for example, that of Suckling, which is identical with the case of Bennett-May of Birmingham, and a second instance is that of Weir, which corresponds with the second case credited to Birdsall.

To the seven completed operations for tumors of the brain which have already been enumerated, must be added: four cases of extirpation of conglomeration of tubercle, the syphiloma of Macewen, the three cases just mentioned which belonged to Heath, Hammond and myself, and finally we must also add the three cases of operations for tumors in which, besides brain symptoms, there were other disturbances, namely: preceding or simultaneous existence of tumors of the same nature in the neck, on the convexity of the skull and in the orbits, these serving as an aid to the diagnosis; thus we can consider eighteen cases in which operations were performed on account of the assumption of the existence of an endocranial tumor. Of these, eight seem to have recovered lastingly. No doubt, in those that recovered, there were remaining defects dependent upon the preceding destruction and atrophy of nerve substance by the tumor. In this category belong the cases of Seguin-Weir (pareses, aphasic disturbances in speech, and epilepsy), Keen (pareses and epileptic attacks), Horsley ("no increase in the previously existing paralysis"). In the two cases operated upon for tubercles, limited epileptic attacks occurred in Horsley's case, while Macewen's patient remained well. In the case of Macewen's which was operated upon for syphiloma, continuously good health followed, as did also in the case of Macewen's operated upon for metastatic sarcoma and in Durante's case of removal of a growth extending from the cranial cavity into the orbit.

Eight cases died as a result of the operation, and two from residuum (Horsley) and extension of the growth (Birdsall-

Weir). The causes of death in the eight fatal cases were: in two cases, meningitis suppurativa (Bennett-Godlee and von Bergmann); of these two cases it may be said that with greater care in bandaging, the infection might have been prevented. In one case death must be attributed to loss of blood resulting from secondary heinorrhage which could not be checked (Birdsall's first case). In the other cases the cause was shock or collapse which terminated fatally in a few hours after the operation, even in the cases in which the tumor was not found (Hammond) or the operation had to be left unfinished (Heath).

Accordingly I am justified in giving the operation only a narrow field and in reserving it for only a few, definitely diagnosticated cases. Even in these cases, its immediate importance is only an exploratory one—that is, an operative procedure subject to further examination. It is only after opening the skull that the operator can be certain whether the new growth is really accessible; and if this be the case, whether its removal is not contra-indicated by its size, its relations to neighboring parts, and its infiltration into these. The surgeon must constantly be prepared to abandon the operation or leave it unfinished sooner or later. With this provision, the attempt to operate upon those tumors which are accessible is allowable; if the surgeon is called before the terminal stage and before a deep coma destroys all hope, such an attempt is even indicated.

I have had an opportunity of admiring the great diagnostic skill of my colleague, Jastrowitz, in a case of tumor of the brain.

This is the case which this observing physician recently published.[79] The history of the case has an important bearing upon the questions which we are discussing. A man, forty-three years old, well situated, father of two healthy children, general health had always been good. Denied syphilis. Since a year and a half had a dull headache in the right side of the frontal region and cranium; this did not lead him to complain, but his pain was inferred rather from hearing him sigh often and finding him putting his hand to his head. Vertigo since November, 1886. End of November, drawing sensations in the left arm and occasional weakness in the left hand. Beginning of December, also weakness in the left leg, increasing

gradually to paralysis, after which there was paresis and then paralysis of the left arm. Ophthalmoscopic examination showed the fundus normal. Sensibility on the left side diminished. Spasms in left arm, leg, and left side of the face, partly with and partly without loss of consciousness, rigidity and contractures in the paralyzed limbs.

When I first saw the patient, middle of January, eight days before his death, he was in a comatose condition. He was under mercurial inunction treatment which produced rapid salivation. When this occurred, it seemed as though he could be aroused at times, at least he answered slowly to questions put to him. But he soon resumed his lethargy. The respiration was of the Cheyne-Stokes type at intervals and later continuously. Regarding the extensive autopsy I refer to the record which is given verbatim by Jastrowitz. Regarding the possibility of an operation, what I have said in the last edition applies here. The large superficially situated mass was covered above by the gray substance of the anterior central convolution. After removal of the roof of the skull, this seemed very markedly swollen, while the posterior central convolution was flattened. There was marked swelling of the commencement of the upper frontal convolution, less of the origin of the middle one. The deeply placed and median mass is situated in the inferior anterior portion of the quadrate lobe, in the angle where the calloso-marginal sulcus turns upward, to just above the corpus callosum into which it extends. No portion of the mass presents any encapsulation. It is difficult to estimate the limits of the new growth on the fresh preparation; after hardening in alcohol it is seen that everywhere it is separated from the lateral ventricle by the distance of 1 cm.

The large tumors which Horsley and Keen removed successfully were encapsulated fibromata or fibro-sarcomata. They could be separated bluntly from the surrounding brain substance which was simply subjected to pressure by them, with the finger, elevator, or handle of the scalpel or of a spoon. It is different with infiltrating tumors: in these cases sharp incisions into the surroundings, which are usually œdematous and the seat of small extravasations, are necessary. These cannot be removed, as far as sight and feeling will guide us, without wounding the ventricles, if, as in the case of Jastro-

witz, the growth extends almost to the roof of the cavity of the brain. Only a small mass of tubercle, scarcely the size of a hazelnut, could be incised in this manner by Horsley, while the infiltrated sarcoma of Seguin's case which was removed with the spoon was of even smaller size.

The operation of cerebral abscess stands in a somewhat opposite relation to that of cerebral tumor. Diagnosis offers too little for the former, but surgical technique is adequate. Regarding tumors, on the other hand, diagnosis has already offered so much that Nothnagel, for instance, looks upon it as a relatively easy one; our technique, however, as far as it applies to an extensive incision and cutting around, control of hemorrhage, and avoidance of dangerous œdema of the brain, is undoubtedly still in the background. Perhaps we may succeed in improving and perfecting it.

I cannot close the chapter on tumors of the brain without pointing out one of the effects of these, which until the present time seems to have been considered too little—I refer to the very peculiar relations of the bones of the skull which is important for the study of cerebral pressure. In the histories of cases, there is often mentioned a tenderness over the situation of the endocranial growth. But the skull also shows another peculiarity at times: a visible thinning-out at certain places, so that at such places it may be as thin as parchment and could be perforated by the finger as easily as could parchment. In a few cases, these thin spots have really become openings and defects. Sonnenburg[80] has described an interesting case of this sort and has drawn the numerous defects in the skull which affected the left side of the frontal bone chiefly. These defects were not due to the tumor growing into the bone and absorbing it. On the contrary, everywhere between skull and tumor there was brain substance: the convolutions, though broad, thin, and stretched, were preserved. There could not, therefore, have been a direct effect upon the bone, but only an indirect pressure effect of the tumor.

In the same way, in eighteen of the cases described by White,[81] there was always a layer of healthy brain substance between the fossæ, the thinned wall of the skull, and the tumor in the brain. In these cases also, the whole bone, frontal, parietal, and occiptal, was not uniformly thinned, but irregularly so. Since direct contact of the tumor could not have

caused the excavation, it can only be explained by increased intracranial pressure. To the extent to which this is due to increase in the tension of the cerebro-spinal fluid, this affects the walls of the grooves and canals which contain it and which communicate with each other in an equal manner. But besides this uniform compression, there is a strictly local effect of this same pressure, which affects certain parts of the organ more than others, or even only affects certain nerves running along the base of the brain. In this manner those indirect area symptoms occurring with cerebral tumors are explained, of which Wernicke writes that, caused by the general compressive effect of the tumor, they have an incalculable connection with the seat of the growth. In the same manner that certain parts of the bone are alone subjected to excavation because they are influenced by the pressure more than the surrounding parts, we may also imagine that it is possible for a favorable direction of growth or a displacement to be the cause of a greater pressure upon one portion of the brain than upon another, and that a distant part may be affected more than a nearer one.

CHAPTER IV.

THE CURE OF EPILEPSY BY TREPHINING.

Scarcely in any branch of surgical statistics have figures been proven so fallacious and valueless as in the treatment of epileptic paroxysms. The more attempts that were made to establish by statistics the beneficial results of trephining in epilepsy, the less have we accomplished and learned from these figures. Recently in four statistical articles collected by MacDougalls[82] it was shown that of 296 cases trephined for epilepsy, not less than 179 recovered; since this report, the operation has lost almost all credit with surgeons. If we examine the histories of a few of the cases reported as cured, it would almost appear as if the authors understood by the word "cure" a favorable result of the operation itself and not a cure of the disease for which the operation was performed. It is certainly surprising that of the 296 cases operated upon, 50 deaths occurred. If we examine the original reports concerning one or more of the cases which were successfully operated upon, we find, especially in the cases of the past few years, where the wounds treated antiseptically healed rapidly, that the patients left the hospital at the end of three or four weeks fully cured and without any return of the epileptic paroxysms. Even if there were some among these cases who had had daily paroxysms before the operation, still the absence of the disease for the three succeeding weeks proves nothing as regards a permanent cure. In institutions for epileptics we can readily satisfy ourselves of the fact, that, at certain seasons, the paroxysms become much less frequent than at others and may even remain absent for a very long time. Besides this, another fact must be taken into consideration; it is well known that a great variety of conditions acting upon the skin and mucous membranes may occasionally cause a disappearance of the epileptic paroxysms for a time. Therefore we can readily understand how it is that every year new remedies are

recommended for this very prevalent disease. In like manner, we can explain the good results, as shown by the collected surgical statistics. I must again call attention here to an article by Maclaren ("Edinb. Medical Journal," 1875, January). Maclaren collected the cases of epilepsy in which major operations had been performed, not to cure the epilepsy, but for the cure of other diseases, such as carcinoma, affections of the joints, etc.; he found that after these operations the paroxysms did not appear for a long time, and in some cases even disappeared entirely. Not long ago, cases were related and described [83] in which epilepsy was reported to have been cured through an accidental burning or a severe cauterization. The majority of communications which refer to the cure of epilepsy by trephining were certainly written before the wound was healed. The operator, happy in the knowledge that two weeks or more had passed without an epileptic paroxysm, felt it his duty *urbi et orbi* to publish his success.

Not unsatisfactory statistics, but the single case which has been watched and followed up for years after the operation and found cured, guides us in investigating conditions under which we may trephine with more success than heretofore.

It is well known that neuropathologists make a distinction between genuine epilepsy, which they are inclined to consider a neurosis, and reflex epilepsy in which the paroxysms are called epileptiform rather than epileptic. I believe that, since Brown-Séquard taught us how to make rabbits epileptic, by wounding their sciatic nerves, we may also call that form of epilepsy which is due to injury of the peripheral nerves a true one, for the reason that the symptoms are the same as those shown by the experiments of Brown-Séquard on the rabbits. Not infrequently, the cicatrix represents a zone capable of producing epilepsy, so that if pressure or other mechanical violence is brought to bear upon this zone, a paroxysm is produced. Just as in the case of the animals experimented upon, we observe in human patients that the paroxysm, at least in a great many cases, which later becomes a general convulsion, commences with twitchings in the muscles supplied by the nerves which have been injured. Finally, a considerable time elapses between the injury or the cicatrization and the commencement of the epileptic paroxysm or rather before it becomes a typical paroxysm. If we are informed that the aura

has its origin in the cicatrix, and if we cure our patients permanently by excising this cicatrix, we must assume that a pathological irritation exists which originates in the injured peripheral nerve and that this irritation, kept up for some time or constantly renewed, acts in such a manner upon the cortex of the cerebrum that the cortex becomes subjected to the same conditions which cause that form of epilepsy called neurotic.

It is impossible for me to state whether "the large number of examples," according to Nothnagel,[84] in which epilepsy was cured through excision of a cicatrix or of a tumor pressing upon the nerve or of an irritating adhesion, etc., refers to temporary cure and not to a lasting and permanent cure.

I believe that I permanently cured a case of epilepsy in a young girl on one occasion, by excising a cicatrix which was attached to the fibula, for I am certain that, for two or three years after the operation, she remained free from any paroxysms, which she had had very frequently before. The cicatrix had been very sensitive at first; later, when the epileptic paroxysms set in, the aura began at this point; and it was sometimes possible to induce a paroxysm even by rubbing the cicatrix. Unfortunately details are wanting in this case. In addition to this case another may be mentioned, upon which an operation was performed in the clinic at Berlin, in 1882. A man, thirty-two years old (Carl B.), was wounded at St. Privat in 1870, by a bullet which became lodged in the region of the left shoulder; after this he became epileptic. The paroxysm began with a distinct feeling in the cicatrix which had formed at the site of exit of the bullet; next came spasms in the muscles of the shoulder, then unconsciousness and the paroxysm. One year later, when examined by the military officer, he stated that the paroyxsms had not occurred; but in 1885, he stated that he was again troubled with spasms of the muscles of the left arm. A cure in this case was, therefore, only problematic. In three other cases upon which I operated and in which I excised the cicatrix, I am certain that no cure resulted; there was not even a temporary success. To this list must also be added a case of removal of a cicatrix from the scalp of a young man twenty years old; the connection between the cicatrix and the epilepsy was only assumed because the paroxysms commenced two, three, or four years

after the injury had been sustained; the aura did not begin in the cicatrix, nor were there any reasons to believe that the cicatricial region was the epileptogenic zone. From these cases, we must draw the conclusion, that the cicatrix should only be excised when it represents an epileptogenic zone, or when the aura starts from this point. If this were the basis of selection of cases, the results would probably be better and more lasting.

It is certain that cases of traumatic epilepsy which proceed from nerves and cicatrices on the neck, trunk, and extremities are very rare. Between their occurrence and that of epilepsy due to injury of the vault of the cranium there is a great disproportion, as was correctly shown by the worthy author of the chapter on this subject in the German sanitary reports.[85] In 63 cases of epileptics among military invalids, 46 had head injuries, and 17 injuries of the trunk and extremities. An estimation by percentage including all cases in which the injuries were not fatal, shows that of 8,985 injuries to the head, 46 were epileptics—0.57 per cent; and of 77,461 injuries to the neck, trunk, and extremities, 17 were epileptics —0.076 per cent.

This disproportion between the frequency of epilepsy following injuries to the large surface represented by the trunk and extremities in comparison with the small surface occupied by the head, compels us to form a distinctly peculiar impression regarding epilepsy resulting from injuries to the skull.

It is not to be understood that there are no cases of so-called reflex epilepsy, which originate in the peripheral nerves of the soft tissues of the head. Most certainly this may be the case, but not more frequently than from any of the peripheral nerves of the rest of the body, and consequently they are rare; at most, this statement might be modified a little as applying to the region of the fifth nerve. Carefully observed cases in which a cicatrix existed in the soft parts covering the skull, and in which there was an epileptogenic zone or the starting-point of a distinct aura, are certainly extremely rare. The examples and reports presented permit an entirely different construction, as I once tried to prove in the publication of Neftel.[86] A student remained in a comatose condition for three days, after a severe blow upon his head; his respirations were irregular and his pulse slow. Although conscious-

ness, ability to move, and power of speech gradually returned, in the second week after the injury he was seized with an epileptic paroxysm. Such attacks were repeated and alternated with an intense headache confined to the right side of the forehead and the right eye. From time to time, there were unendurable exacerbations of these pains, lasting for weeks with only short intermissions, and in the intervals disappeared entirely; but they could always be brought on by a moderate amount of pressure upon the portion of the forehead just mentioned. If the pressure was increased the severity of the pain was greatly intensified, and the patient became unconscious and had an epileptic paroxysm. In this case there was certainly an epileptogenic zone, but there was also a severe concussion of the brain, shortly after which (as early as the second week) the epilepsy began. On this account and on account of the character of the injury, we cannot differ with those who consider the case as one of undoubted affection of the convulsive centre in the pons, nor can we differ with those who consider it a cortical lesion, for the cortex may have been injured under the seat of violence without the occurrence of any fracture or wound of the skin. In this account it is still a question whether, in this case, the seat of the trouble was in the peripheral branches of the first portion of the fifth nerve, or the cerebral cortex, or finally the region of the pons at the base of the brain.

Even though, in spite of all these considerations, we are willing to admit the existence of reflex epilepsy due to a lesion of the nerves in the soft tissues covering the skull, still the largest proportion and even the very largest proportion of cases of epilepsy resulting after injuries to the head must be considered as due to other causes.

Our consideration of epilepsy as a result of head injuries must be dependent upon experiences which we have gone through concerning the origin of epilepsy resulting from cortical lesions. These are as interesting as they are important and have been explained and described so clearly that they are gradually eliminating more and more the differences between symptomatic and genuine epilepsy which were formerly so readily applied.

I must remain upon this subject a moment longer, even at the risk of repeating what has been well known long ago.

Since the experiments of Hitzig and Fritsch have established the fact, that irritation of the cortex may produce epileptic paroxysms, a great number of experiments have been made upon animals in order to discover the epileptogenic characters of the cortex. The most important, namely, those of Luciani, Franck, Pitres, Unverricht, Rosebach, Johannson, and others, all agree that in experimenting by means of electrical irritation epileptic paroxysms can only be produced through the influence of the so-called motor area of the cortex.

It is quite true that an epileptic attack can be brought on by electrical irritation of any portion of the cortex, including the frontal and occipital convolutions. But, while upon all other portions comparatively severe blows and long-continued applications must be made, the motor region requires only the very weakest irritation to produce the same effect. The experiments of Unverricht[87] have cast a doubt upon the correctness of the teachings of Rosenbach and others, that after destruction of the motor centres, irritation of the undisturbed occipital convolutions produces no paroxysms, though these experiments support the fact that the motor area is the most important space in the production of epileptic attacks. Under no circumstances is it possible to bring on epileptic attacks in animals when the cortex has been separated from the white substance of the brain.

Consequently we must search for the anatomical seat of the disorders which cause the epileptic paroxysm in those portions of the cortex in which the motor centres lie, especially in the absolute cortical areas, but also in the relative areas. The neurosis epilepsy would be represented as an affection of the cortex, and more especially of a distinct region of the cortex, the motor region. The nature of this affection is still a question, but the cases are increasing in number, in which in the zone under consideration, whether in the substance of the brain itself or in the membranes which cover it, anatomical changes were found in epileptics; that is, in those who became epileptic after an injury to the skull with or without bleeding.

As a matter of fact, in most cases of severe, but especially of localized injuries to the head, the cortex which is affected is that portion which is especially exposed and which, consequently, is most frequently affected at the same time. If we merely considered simultaneous wounding of the brain in cases

of compound fractures of the skull, or of comminuted fractures, we would merely be considering the minority of the cases of contusions of the cortical areas; oftener than we imagine the cortex is also wounded, in cases in which a severe force is brought to bear upon the top of the skull. The reason that this fact has escaped the attention of observers and is still overlooked is, that cortical lesions only produce special area symptoms when they occur within a limited portion of the surface of the brain; if they occur upon any other portions, no symptoms are produced.

A more careful insight into the mechanism of wounds of the vault of the cranium, especially of those effects of force which involve a comparatively small area of the superficial surface, has taught us that in these cases the cortex must necessarily suffer. Thus a shot which strikes the bone and grazes it, whether it grooves it or leaves it intact, whether it strikes vertically or at a tangent, must injure the cortex, owing to the form and elasticity of the skull. Of course, I am not referring to perforating gunshot wounds of the skull, but to those spent bullets which strike the head at the end of their journey. The rapidity of their movement is always sufficient, in striking against the wall of the skull, to strain this, make it level at first and then push it inward and depress it. The elasticity of the bone prevents the flattening and depression from causing a solution of continuity, for the depressed portion of the bone rebounds into its former position. As a result, there is no trace of either a fissure or flaw on the surface, but more deeply, traces of the violence of the blow can be found; that is, beneath the portion of the skull which was struck, limited changes in form will be found, for the brain substance and the arachnoid have entirely different relative elasticities than the bony portions of the roof of the skull. I need only call attention to an experiment which can easily be repeated. Felizet filled a skull with melted paraffin, allowed the paraffin to harden, and then threw the skull upon the ground from a slight height. He then sawed off the roof of the skull and inspected the upper surface of the paraffin. This showed a flattening or a cup-shaped depression at the spot upon which it had fallen. What applies to cases of spent bullets also holds good in cases of violence upon circumscribed areas of the skull, from blows of a hammer, from missiles,

from falls upon or stumbling against any pointed object. Finally we must certainly also think of a complicating affection of the cortex in those cases where the bone has been fractured either as a result of a bullet, a hit, or a fall, that is, where a limited, narrow fracture exists over a small portion of the roof of the skull.

An examination as to which injuries of the soft parts or of the skull cause epileptic paroxysms shows that practically all the cases belong to the category mentioned above. Cicatrices which are adherent to the bone, indurations, inequalities, osteophytes, depressions and flattening of the skull under the cicatrix may all, just as in the case of defect in the bone where the membranes covering the opening are adherent to the skin and to the surface of the brain, be considered as original injuries to the cortex. At the very least, during the time in which the force acted, the cortex was certainly torn, bruised, or altered in some way. A good many of these changes will probably be overlooked in making autopsies; but that they really exist is proven by the peculiar retrogressive metamorphoses in the ganglion cells of the cortex which were discovered by Virchow [88] in those situations where violence had previously been applied to the skull.

We notice experimental physiologists and pathologists seeking the seat of the epilepsy in the cortex; and we invariably find the cortex affected in all those cases in which epilepsy follows an injury to the skull. Is it not necessary, therefore, to consider the traumatic form of epilepsy following head injuries as cortical epilepsy?

I do not wish to enter into the discussion of the neuropathologists, whether every epilepsy has its origin in the cortex and hence, to a certain extent, its anatomical seat here, or whether it is better to consider the convulsive centre of Nothnagel, behind the pons, as responsible for the disease; but I must emphasize the fact, that in those cases of epilepsy due to traumatism, the cortex is affected; and this fact permits us to consider that form of epilepsy following head injuries as due to a special and different cause from the forms of reflex epilepsy which are due to peripheral causes.

Horsley is entitled to the credit of having practically applied this view of the origin of epilepsy to its treatment.

Even before modern physiologists experimented upon ani-

mals in demonstrating the functions of the cortex, Hughlings Jackson described a special form of epilepsy connected with a diseased condition of the cortex; this was named after him, Jacksonian cortical epilepsy.

The experiments made by electrically irritating the cortex have shown us that strong currents and blows produce general epileptic paroxysms and sudden spasms in all the muscles of the extremities. A weaker irritation of the real epileptogenic centres, that is, of the motor cortical centres, gives us, on the other hand, a definite development and extension of the clonic spasms, always occurring in the same manner.

All observers acknowledge and confirm that which Ferrier and Luciani first discovered, that the paroxysm always begins in the group of muscles which corresponds to the centre in the cortex which has been electrically irritated, that is, the centre which belongs to this group of muscles and which presides over it. Sometimes the spasm is limited to one group of muscles (monospasm); at other times it spreads to the muscles of one half of the body, or still further over the entire body (hemispasm and general epilepsy). Every propagation from the centre which was originally irritated has its own distinct course and this course is always the same. This course is governed by the anatomical arrangement of the cortical areas; none are skipped, the irritation always extending from one area to the adjoining one, then to the next, and so on. If, for example, we irritate the centre on the left side which presides over the movements of the eyelids in a dog, there will first be spasms of the right orbicularis palpebrarum, next of the levator anguli oris, next convulsive movements of the muscles of the ear, then nystagmus, opening and closing of the jaw, and a spasm of the muscles of the tongue; then the head will be turned toward the right side and spasms commence first in the upper and then in the lower extremity; all this corresponds exactly to the positions shown by the drawings of Ferrier, which illustrate the cortical centres in the dog, extending continuously from one centre to the next; the muscles which correspond to adjoining cortical centres begin to twitch in succession.

Not only the opposite side, but also the same side of the body gradually becomes affected with the convulsive movements. And here, too, a well-regulated type exists. After the con-

vulsion has spread over all the muscles of the opposite side, it gradually affects the same side, always ascending, beginning in the lower extremities and then extending to the groups of muscles of the upper portion of the body. If we irritate the centre for movements of the muscles of the eye in the left hemisphere, as was shown in the example just cited, the convulsion starts on the right side, commencing above and extending downward; after the convulsion has extended to the left side, the reverse is the case; for the muscles of the lid are not the first to become affected, but the last, the paroxysm commencing in the lower extremity, spreading to the upper, and continuing until it becomes general. To a certain extent, the convulsive movements pass around the body; commencing in the muscles of the lid of the right eye, they continue downward and extend to the right leg and foot, thence to the left side, extending upward from the foot to the leg, and so on until they reach the muscles of the left lid. The progress of the proxysm is much slower in the variety of epilepsy due to cortical irritation than in other forms. The symptoms follow each other much less rapidly and on this account we are clinically able to follow their course.

It is obvious that regularity in the commencement and course of the spasms is an important factor in the diagnosis of the origin of the disturbance. Of equal importance is an observation made by Unverricht regarding the beginning of the epileptic condition in animals experimented upon; after the first paroxysm is over, and it is rapidly followed by a second, third and fourth, the succeeding ones always have the same point of origin, *i.e.*, from the same group of muscles which initiated the first paroxysm.

In short, those epileptic paroxysms which are due to any disease or any affection of a limited and circumscribed area of the motor cortical zone, manifest so many special and peculiar factors that they may be recognized as such, *i.e.*, as results of an irritative condition affecting a small but definite portion of the cortex. This area must be in such a condition of excitation as to initiate an epileptic paroxysm. We are still unable to say, by what anatomical changes these conditions of excitation can be recognized; but a number of macroscopical changes in the vicinity of the affected areas are exposed, sometimes by the surgeon's knife, at other times by that of

the anatomist. Excluding changes in the bones, such gross lesions include: adhesions between the membranes covering the motor area; gross-meshed, cystoid scars filling up flat, cup-shaped depressions on the surface of the brain; rusty-colored, thin connective-tissue layers and thick sclerosed and dense plaques in and on the cortical layer; and finally, splinters of the internal table which have become incorporated with the tissues underneath or penetrating foreign bodies of all kinds, complicating injuries.

Unverricht's attempt, which was based upon a series of experiments in which the results agreed, and which sought to establish a definite, clinical picture in the cases of epilepsy emanating from the cortical areas of the occipital region so as to make the diagnosis of this variety of epilepsy possible, is worthy of notice.[89]

Broca has shown that attempts were made to cure epilepsy by boring or chiselling away a piece of the skull as a cure for the *morbus sacer*, as far back as the prehistoric times; this was looked upon as an operation which sanctified the one operated upon. It is possible, therefore, that the trephining of epileptics is the oldest of all operations, and that a description of its efficacy is an inheritance from the stone age. As trephining became more limited in its application, as gradually brought about from the time of Desault to that of Dieffenbach, its employment in epilepsy gradually became less frequent until in the last three or four decades cases were only trephined when a cicatrix existed upon the skull, or any other mark was found, produced by the varieties of traumatic injuries which have been enumerated above. Whether by the inauguration of antisepsis and the protection which this insures, trephining has been resorted to more frequently in these cases of traumatic epilepsy, I cannot say. Seydel's compilation[90] would not lead us to believe this; for he was able to collect only twenty-nine cases which had been trephined for epilepsy since the introduction of antiseptics. This small number of cases may be explained by the failures concerning the latter; at least concerning those which occurred in my practice in Berlin, I am able to add some observations:

The first case occurred in a landowner from Silesia, who was wounded in the war of 1870–71; I do not find any mention of his case in the sanitary reports of the medical division

of our war department. He was in the field with the 5th corps of the army and was wounded at Champigny, December, 1870; he was then brought to Ludwigsburg, near Stuttgart, by a sanitary train. In this place a number of fragments of bone were removed from the depths of a grooved gunshot wound of the left parietal bone, and finally the wound healed. Two years later, the first epileptic seizure occurred. The attacks first came at long intervals, then more frequently, and during the last year several times a day. As his memory began to fail and he became weak mentally, his family consulted our neurologist, Dr. Westphal, who called me into consultation and proposed trephining the injured portion of the skull. The injury which had necessitated the extraction of fractured particles of bone seemed to warrant the supposition that a necrotic process had existed at the injured spot and that thickening of the bone and dura were present. There was nothing peculiar about the convulsions; from the beginning, there were spasms of the head, trunk, and extremities. January 15th, 1884, I trephined. The bone was chiselled away over the entire extent of the scar which ran across the parietal eminence from before backward; it showed no trace of previous injury or of any defect and the dura was smooth and shining and not more adherent to the bone than normal. The wound was closed immediately and healed in a week under a bandage. The epileptic attacks, which had occurred daily and even four times a day before the operation, now ceased; this continued until February 16th, a full month. The patient had already left the clinic and was preparing for his journey home, when, on the evening of the day mentioned, he was seized with a very severe convulsion, followed rapidly by a second and a third. An epileptic state developed in which the patient died. The autopsy showed the healing of the wound and integrity of the dura and of the cortex of the brain situated beneath the seat of trephining. There were no changes in the brain worthy of mention.

The second case occurred in a boy of nine, who had received a complicated fracture of the skull in the right parietal region as a result of falling out of the window when four years old. After two years the first epileptic convulsions began, at first seldom and then more frequently. In this case also, there were typical, general convulsions with loss of consciousness.

The trephine was applied over the seat of a distinct depression in the bone. The attacks which had occurred daily before the operation, did not occur for sixteen days after the operation. Then a seizure occurred, a week later another followed, and so on.

The third case of trephining had a similar course; it occurred in a man twenty-six years of age. During the first week after the operation there were no seizures; then they recurred just as they had been before the operation.

A boy, eight years old, who had fallen upon his head several years before and who seemed to have a slight unevenness of the bone in the region of the injury, began to be epileptic three years after the fall. I trephined him because I found this small groove. The bone was healthy, but on the inner surface of the button of bone that had been removed with the trephine, a yellow spot, a small velvety osteophyte was found. Rapid healing beneath a bandage. An attack three weeks after. The attacks were less frequent than formerly, but had the same general characters. I put him immediately upon large doses of bromide of potassium. The attacks disappeared for half a year and then they returned.

A very strong man of twenty-four had had his skull smashed in about the centre of the left half of the frontal bone, by the falling of a stone, eight years before. Here there was a depression into which a hen's egg could almost be placed. There were epileptic attacks almost every week. The depressed portion of bone was chiselled away and was found enormously thickened. After a rapid healing of the wound, the seizures remained absent for four weeks. Then the patient died from poisoning from illuminating gas, having gone to bed intoxicated after having blown out the gas without turning it off. This patient therefore, remained cured until the time of his death!

It is not the antiseptic era, but the new direction which Horsley has given the question of trephining in cases of traumatic epilepsy that seems to me to offer better prospects in the operative treatment of this common disease. And again, such success certainly cannot be expected if we trephine every case of traumatic epilepsy after injuries to the head, but only if we limit this operation to certain very distinct and well characterized cases; in other words, if we look for success in the quality and not in the quantity of our cases.

In order to understand this new indication, we must proceed from those experiences of experiments and practice which place the *causa et sedes morbi* in the cortex of the brain, and make it a substratum for the disturbance which initiates the convulsion. Consequently, it is necessary, as Horsley has demonstrated, to extirpate as thoroughly as possible all that which we find pathologically changed in this part as well as in others. While formerly the operation was limited to the removal of skin and bone cicatrices, or at most included the separation of the dura from the bone in cases in which these two had become adherent, Horsley has added a further task: the operation should always include the opening of the sac of the dura, and thus lead the way to the diseased areas which are to be removed *in toto.*

But in order to proceed in this manner, it is necessary for us to confine our operations to those cases in which we have good cause for assuming the existence of a real and therefore a removable lesion of the cortex. However, such an indication only exists in those cases in which the convulsions begin, extend, and become general, exactly as in the irritation experiments upon animals.

Although Wiesmann describes a successful case of trephining in the case of an epileptic, in which the dura was incised and the highly œdematous pia was cut into, in his large work on the modern indications for trephining,[91] still the subject of the methodical removal of cicatrices from the surface of the brain was first brought to the attention of surgeons and first made to interest them by Horsley. Since German periodicals have given very little in reference to Horsley's observation, I take the liberty of giving an exact translation of this important case:

Jacob B., aged twenty-two, was admitted to the National Hospital and placed under the care of Drs. Hughlings Jackson and Ferrier. When seven years of age, he was run over by a cab, in Edinburgh. At that time, a comminuted fracture with depression accompanied by the loss of brain substance was diagnosed by Dr. Annandale in the Royal Hospital. The fragments of bone were removed at once; the healing of the wound took considerable time, however, since there was much suppuration and the development of a hernia cerebri.

The patient was hemiplegic for a time, but the paralysis disappeared after seven weeks. When fifteen years old, intermittent epileptic seizures appeared. In 1885, upon admission to the hospital, one attack followed another, so that for several days he was in an epileptic state. After his discharge, he was free from attacks for seven weeks, then they reappeared, and three days before admission he was again in an epileptic state.

Present condition.—On the left side of the median line, there was a scar; in the centre of this scar an oval opening in the bone could be felt which was about one inch long and which ran in a direction corresponding to that of the sagittal suture. The exact seat of the scar corresponded to the middle of the upper third of the ascending frontal convolution, as determined by measurements; that is, it lay behind the posterior end of the upper frontal fissure. Pressure upon this scar caused pain, especially when the patient had attacks. The seizures, which occured in great numbers—the patient had no less than 3,000 of them in two weeks—always had the same character: they began most often in the right lower extremity, sometimes at the same time also in the right upper extremity. The following is an example of an attack of the first category: the right leg was first extended tonically, before it was affected with clonic spasms. Then the right arm was slowly extended, until it formed a right angle with the trunk, while the fingers and the wrist were flexed. After this there was extension of the fingers, followed immediately by clonic spasms of the whole extremity, terminating with flexion at the elbow. The spasms of the lower extremity ceased in the mean while; while the twitchings of the fingers, hand, and arm continued, the right angle of the mouth became involved and subsequently the entire right half of the face. Finally the head and the eyes were turned to the right. Consequently the succession in the involvement of the affected parts was the following: Lower extremity, upper extremity, face, neck. The character of the movements was the following: First, extension, then convulsions, and at last flexion. This gave a distinct indication of where the diseased area was to be sought for—namely at the posterior end of the upper frontal sulcus. The result of the measurements over the situation of the scar corresponded with this diagnosis. After the attacks, and even for ten days

after the last attack, the patient seemed to be hemiparetic. The movements executed with the right side possessed but one-half the strength of those of the left side. Sensibility was not altered on the right side; reflexes, superficial as well as deep, were exaggerated upon both sides.

Operation on the 25th of May, 1886. The bone was removed around the opening in the skull already described, antiseptic precautions being observed. The dura mater, arachnoid, and integument formed a homogeneous mass of fibrous tissue which was turned back with the skin flap. The cicatrix in the brain was very vascular, of a deep red color and about 3 cm. long and 2 cm. broad. The pia covering it seemed opaque and the surrounding portion of the brain had a yellow color. The scar was removed and with it the surrounding substance of the brain for a distance of about one-half a centimetre, the incision extending to a depth of 2 cm. It was then discovered that the scar had penetrated to the distance of several millimetres into the fibres of the corona radiata of the marginal convolution; this portion of these fibres was removed and the wound was then closed. In doing this it was necessary to tie three pretty large veins which seemed to come from what had been determined as the cortical area for the upper extremity. The wound healed completely in a week.

It was highly interesting to observe that immediately after the operation the fingers of the right extremity were paralyzed, and it was also impossible for the patient to flex the hand and to supinate his forearm. With this motor paralysis, there was loss of sensibility (tactile) over the dorsal surface of the fingers. The patient was not able to locate touch on the under surface of the wrist-joint, nor was he able to indicate the position in which the fingers were placed. This was undoubtedly, then, an example of loss of sensibility and of muscular sense, associated with motor paralysis. In this connection, we must not forget that probably a few fibres which passed from the gyrus fornicatus into the corona radiata had been injured. The condition of motor and sensory paralysis disappeared in the course of the following two months. The epileptic attacks have disappeared and had not returned up to the time of the meeting of the British Medical Association, in August.

Horsley's second case, which was published in 1886, occurred

in a man of twenty-four, who had sustained a complicated comminuted fracture near the parietal eminence during his fifth year. Eight years later he was injured in the same situation by the kick of a horse. The epileptic attacks began three months after the last accident, three or four successive attacks occurring at intervals of three weeks. Upon admission to the hospital a scar was found over a depressed area in the upper anterior angle of the left parietal bone, extending to the middle line. The cicatrix was tender to pressure; the frequent and severe headaches of the patient were also referred to this region. Tenderness upon pressure and headaches were regularly increased at the time of the paroxysms. There was complete right-sided hemianæsthesia. In this situation, the patient felt neither touch nor the point of the needle; however, the application of the faradic current caused this disturbance to disappear, while the seizures continued. The latter commenced with an aura proceeding from the abdominal organs. The patient had desire to defecate, and pain in the left side of the abdomen. After this there was a feeling of constriction in the throat and occasionally a cough. Then his head and eyes turned toward the right; the right arm was pushed out backward and then only the loss of consciousness resulted. Then followed violent convulsive movements of all parts and these were of a rotary character. The lower extremities were extended. After the attack there was weakness in the right arm for some time. The centres for the muscles of the abdomen are situated upon the surface of the gyrus marginatus, just opposite the posterior extremity of the upper frontal convolution, while, according to Ferrier, the cortical area for movements of the head and neck lies beneath the first frontal sulcus and that for extension of the arm lies above the posterior extremity of this sulcus. On this account, Horsley diagnosed the seat of the irritating disturbance in the posterior third, that is the root, of the upper, large frontal convolution—a spot which corresponded exactly with the seat of the depression upon the skull. Operation, July 13th, 1886. Formation of flaps from the soft parts and removal of all the depressed bone; then the former fracture of the internal table could be demonstrated. The dura covered in a small, narrow plate of bone which occupied an excavation of the brain. This fairly-marked cavity was filled with a clear fluid and with spongy,

loose connective tissue. It occupied the situation which the diagnosis had considered the seat of the irritation. The entire cavity was excised by an incision extending around its entire circumference at a distance of about 5 mm. The wound healed in four days. One week after the operation, the patient complained of a weakness in the entire right upper extremity which involved the hand especially. In a little over a month, this paresis had disappeared. After the operation, there were three insignificant attacks; after this, there were none.

In his table of April, 1887, Horsley briefly narrates two other, analogous cases:

A man, thirty-seven years old, was injured fourteen years previously by a blow upon the head. Three years after this injury he had his first epileptic attack. On the left side a scar could be seen corresponding to the upper extremity of the fissure of Rolando. There was motor and sensory paresis of the right side, most marked in the big toe of the right side. The patient is indifferent, stupid, and his memory is weak. November 8th, 1886, trephining. The bone was found healthy. After incising the dura, a large cystoid scar was removed from the cortex of the brain. Lister dressing and healing by first intention. The paresis of the upper extremity improved, that of the lower remained unchanged. Sensibility had increased visibly. On the day after the operation he had four attacks, and six days after the operation, he had two. The attacks were less severe and were not accompanied by any loss of consciousness.

The last case occurred in a child four years old, who had from to three to fourteen attacks every day. The right side of the body was completely paralyzed. From time to time he fell into a semi-comatose condition. The diagnosis was a hemorrhagic (cystoid ?) scar in the motor region of the left hemisphere. Trephining, November 29th, 1886. Exploration of the brain by an incision. Strict Listerian treatment. No drainage-tube, but the lower end of the wound was allowed to remain open. The condition of the paralysis and of health in general remained the same as before the operation. No attacks for a month; then six slight ones; after this no further seizures.

The short time of observation, until April, 1887, is not decisive in regard to how permanent the improvement in these cases was.

In all of his cases, Horsley removed larger or smaller pieces of the cortex of the brain with the cicatrix or the traumatic cyst beneath the dura mater. One's first impression would be that these extirpations of portions of the cortex of the brain would be accompanied by a good deal of risk. But what we know concerning the course of paralyses which result after removal of cortical areas, justifies Horsley's proceedings. The functions which have been lost through the injury and disease of pieces and portions of the cortex of the brain, reappear in time, since, as we believe, other portions of the cortex or even provinces placed more deeply, take their place vicariously. If the therapeutic effect depends upon the complete removal of the scar, the excision must be done regardlessly.

During the past years not a few operations of this sort have been performed. Only a few especially instructive cases will be cited here. Denous (Congrès Français de Chirurgie, 1883, p. 308) operated upon a mechanic who had severe cerebral manifestations in consequence of a fall into a well, and who suffered from paralysis of the left upper and the right lower extremities, since many months. A pretty complete cure was effected except that a slight paresis of the right leg, loss of tactile sensibility in the left hand, and a decrease of hearing on this side resulted. Two years later there was a sudden repetition of the epileptic seizures, limited to the lower branches of the facial and to the extremities of the left side; these seizures were followed by paralysis of the left side. There were no changes on the right side of the skull; but on the left side there was a scar somewhat behind the parietal eminence, and with it a depression in the bone. The operator ignored this scar and the old paresis of the right leg and applied the trephine on the right side about the centre of the fissure of Rolando. At this point the remains of a linear fracture of the skull, 3 cm. long, were seen; after removal of the dura beneath this spot, a meningo-encephalitic collection was found. "La section de la dure-mère est indispensable, on le comprend, pour arriver sur le foyer du mal et même l'ablation de la portion altérée de l'écorce cérébrale peut être utile pour arrêter les accidents qui menace la vie." A slight attack occurred several hours after the operation; but none after this. Several months after, the wound had healed and the paralysis of the

left side had disappeared. The old disturbances in the right leg, left hand, and in the brain had remained unchanged. At the time when he was presented, twenty-three months after the operation, the patient was still free from epilepsy.

Macewen (l. c.) reports the case of a patient, twenty-two years old, who suffered from epileptic attacks; these occurred on the average once every five minutes, so that in the course of the day he had almost a hundred seizures. The spasms were confined to the tongue, the muscles of the face, and the platysma of the right side. When the twitchings ceased, the affected parts remained paretic. Consciousness was always preserved during the attacks. Eight years before, the patient had received an injury to the skull, after which his right arm became paretic without interfering with his work. At the operation, a cyst about the size of a hazelnut was found at the base of the anterior central convolution; this cyst was situated partly in the cortex and partly in the white substance of the brain; around it, encephalitis had developed. During the enucleation of the cyst, the patient, though in deep chloroform anæsthesia, had a seizure which was limited to the same groups of muscles which had become affected before the operation. The removal of the cyst determined the cessation of the attacks; and since that time they have not occurred again. The wound healed rapidly. The right facial paralysis disappeared rapidly and the right arm also grew stronger. This case of Macewen's is probably the most interesting and important among all the cases operated upon for lesions of the cortex, not only on account of the lasting cure of the seizures, but also on account of the type of the convulsions which corresponded exactly with that observed in experiments upon animals. On this account it was also the means of establishing the seat of the facio-hypoglossal centre in man for the first time, and of demonstrating that this corresponds exactly with the position of the latter in the dog and ape.

Park[92] treated a girl of fourteen, who had received a contusion of the right side of the head in falling out of a window; she was unconscious for ten days and then suffered from hemiplegia which affected the left side, and which disappeared gradually. Two years later, she had an epileptic seizure beginning in the right arm and extending over the right side of the body. A depression in the bone was found one and one-half

inches above the left ear. The diagnosis made was a cyst produced by an extravasation of blood over the centre for the right arm. Operation, February 24th, 1888. The opening in the bone was of large size, measuring 2½ cm. in diameter. Beneath this opening, there were fibrous laminæ adherent to bone and dura and between the layers of these a cyst was found. The bony opening was enlarged, and a cyst 3½ inches in diameter was opened into and emptied; at the same time, the portion of the dura which covered it was cut away. Rapid primary union. Decided improvement. However, subsequently, when she suffered from digestive disturbances, the epileptic attacks recurred.

A second case, also reported by Parks, is similar to the preceding: A man of forty-six had fallen from a wagon and had been unconscious for sixteen hours after; he had slight paresis of the right leg, paralysis of the right, and motor aphasia. Four months later he had his first attack of epilepsy; then others followed in frequent succession. The operation of opening the skull, November 17th, 1886, had neither a depression nor a scar as a guide. The dura bulged into the wound. Underneath, a cystic tumor containing 1½ ounces of clear fluid and having the dimension of 5×2×2 cm. was found. Drainage of the wound; rapid healing. Excepting the disturbance of speech, marked improvement followed.

At the reunion of Berlin surgeons, Langenbuch [93] presented a girl of about six, who had fallen out of bed upon her head, when she was three years old. As early as the following night, she had left-sided convulsions of the extremities and the face. Then for two years she was perfectly well; after this, the convulsions returned and recurred at intervals of five or six weeks; they had the same character as before. They always commenced in the left hand, then extended into the left arm, the face, etc., all this occurring slowly so that it took two or three hours. Supposing the existence of some sort of permanent, traumatic lesion of the cortical areas around the fissure of Rolando of the right side, Langenbuch exposed this region. The tense dura appeared in the trephined opening, devoid of pulsations. This tension was caused by a cyst in the arachnoidal tissue, about the size of a walnut, which was opened and emptied. Uninterrupted healing. For a longer period than ever before, sixteen months, the child

had no paroxysms; then an attack resembling the former ones occurred.

Salzer,[94] de Arreilza,[95] and others have operated in the same manner.

The examples made use of, unfortunately, only show imperfect results. Only in the case in which there existed a real, typical Jacksonian form of the disease and the spasms remained confined to few groups of muscles, in the case of Macewen, was a lasting result—a cessation even at the end of twenty-three months—reported. In the other cases, even though the palpable changes, cysts or cystic degenerated portions of the pia mater and cortex of the brain were removed, a cure did not result.

The possibility of removing a limited and small portion of the cortex of the brain without lasting injury is associated with another consideration in relation to the checking or cure of epileptic attacks. In cases in which the epilepsy starts from one and the same cortical centre, does it not seem as though the excision of this cortical area ought to act curatively, because, taking for a basis the observations just mentioned, we may, to a certain extent, regard it as the substratum of the disease?

There is much in connection with experiments on animals to support this idea. Munk and also Heidenhain succeeded in checking convulsions of the opposite side, by rapidly extirpating the whole motor zone of one side. Novi confirmed these experiences, in Luciani's laboratory. During convulsions in a dog, a rapid incision with the knife into both gyri sigmoides will, *ipso facto,* stop every movement of the extremities; only rhythmical motions of the muscles of the face and lower jaw are preserved; however, these also disappear if the incision be continued outward and downward from the gyrus sigmoides to the areas which control motion in these muscles.

In one instance which was not a clean enough case to make it valuable as a test, I endeavored to cure epilepsy by extirpating a definite cortical area.

The case occurred in a lad of twenty who came from a healthy family free from neuroses; he was born healthy and grew up well until his fourth year. Then (that is, sixteen years ago) he received a fracture of the skull on the left side

as the result of the falling of a large block of wood upon his head. The child fell down unconscious, but recovered so rapidly that he was able to resume his play in two hours. A doctor who had been summoned diagnosed a complicated fracture of the skull, and cured it after suppuration lasting several months. Of the special symptoms we were informed only that after the injury he had had a paralysis of the entire right side of the body which disappeared, however, before the wound had healed.

Two years later, in his sixth year, he became subject to attacks of vertigo which did not, however, prevent him from attending school. He is reported to have advanced in school; but the teacher noticed his lazy and indifferent disposition. Later, it was observed that he suffered from convulsions; he became red in the face, threw himself to the ground with a cry, and became violent; it was said by those who saw him, that his right side was contracted and that he lost consciousness. After a time, the seizures occurred more frequently until as many as six occurred on one day. There were more favorable periods, but rarely; however, once, after having been treated with bromide of potassium, he was free from attacks for a week.

Shortly before he was brought to the Royal Clinic, he had passed through a very severe period of almost continuous attacks. His mental capacity had suffered markedly of late; a weakness in his right arm and leg was also noticed, so that he scarcely walked, and when persuaded to walk he dragged the right leg after him.

The patient gives an impression of being very deficient mentally. Most of the time he lies in bed and stares in front of him in a vacant manner. When questioned, he gives correct and distinct answers, but he does not engage in any conversation with the other patients. No disturbances of any of the organs of sense. Appetite is excellent; bowels regular.

He is of small stature, but well-nourished. Fat and muscles are developed beautifully, except that the right arm and leg are thinner than their fellows. The movements of the right extremities are free, but occur spasmodically. Since he does not usually succeed with the right hand, he prefers using the left. The right hand is always extended, as are also the fingers. They can be flexed and extended, both actively and

passively; but when at rest, they assume the extended position. If the hand be grasped and an attempt made to flex it, we feel an unexpected resistance. In the same way the flexor muscles of the forearm become rigid upon attempting to flex this portion of the upper extremity.

The convulsive attacks occurred at least twice a day and on this account could be observed accurately. Without exception, they began with over-extension of the hand and fingers of the right side. Then followed twitchings of the whole arm and later of the right lower extremity, finally the whole body becoming involved. At times the general convulsions followed immediately upon the tonic hyperextension of the right hand. Toward the end of the attack, the head was usually turned to the right side.

Not all the attacks had the same characters; there was an alternation of severe attacks with slight ones, of lengthy with short ones, of general clonic convulsions with others which remained confined to the right side and consisted of merely a few twitchings and convulsive movements of the body.

One thing was, however, constant—at the beginning of every attack, no matter what its character was, the right hand was in a condition of tetanic extension. The examination of sensibility was unsuccessful since the patient gave answers which were too indefinite. On the left parietal bone of the patient, there was a white, smooth scar, but little depressed, the centre of which was about the size of a quarter and sent out three prolongations forward, upward, and downward. The scar was adherent to the bone beneath, which showed a small, sharp-rimmed opening. If, with Broca's flexible, angular measure, we first determine the point of junction of the coronal and sagittal sutures, and proceed backward 45 mm. in the median line, the side of an angle of 70° proceeding from this median point will meet the anterior portion of the central part of the scar. In addition, it was determined that the deepest part of the cicatrix corresponded to midway between the semicircular line and the sagittal suture. Consequently, we were justified in assuming that it covered that of the psychomotor centres in which the movements of the hand especially have their seat.

Thinking of the experiments which were to establish the connection between the scar and the cortical areas of the

motor region, I invited Prof. Munk to the operation; and I take this occasion to thank him for the assistance and interest which he gave our patient.

February 2d, 1887, I trephined in the situation already described. In pushing back the periosteum, an opening in the bone having a width of 3 to 5 mm. and a little greater length, was exposed. The scar in the skin and the dura and arachnoid had become agglutinated, so that in exposing this opening, there was an escape of considerable cerebro-spinal fluid. After exposing the skull freely, I chiselled away about 5 cm. of bone all around the margins of the opening. In removing this, a fragment of fractured bone was found, close to the scar; it was somewhat smaller than a silver quarter, and had become placed beneath the roof, between the dura and bone. The upper margin of the removed portion of bone ran parallel to the sagittal suture, but certainly 1 cm. distant, so as to avoid the longitudinal sinus. Although hemorrhage was slight, every bleeding point was taken into account and controlled by twisting or ligature with catgut; when hemorrhage had ceased completely, and the wound had been irrigated with 1 to 2,000 corrosive sublimate solution, the operation was continued. I incised the dura by a circular incision extending all around the circumference of the opening in the bone and removed it, again securing every bleeding vessel. In the situation of the scar, the adhesion was firm. The surface of the brain presented a deep depression in this situation; this was filled with a reticular tissue permeated by cystoid spaces, from which, apparently, the fluid which escaped during the operation, had come. I was also compelled to grasp the pia mater with a pair of forceps and pull it off before I could locate the positions of fissures and convolutions. Then the fissure of Rolando showed plainly; but on the surface of the brain in this situation there was neither discoloration nor any other change. I extirpated a piece of the surface of the brain of about 1 c.cm. in extent and 3 mm. in depth, from just behind the fissure of Rolando, at a point corresponding in my illustration [96] to the extensor of the hand (anterior half of the cortical area, 2 *d*).

Of late, as often as I have had to expose bruised portions of brain in trephining, I have refrained from closing the wound immediately and have not put in sutures until twenty-four or even forty-eight hours afterward. This case was treated

similarly. The wound was irrigated gently with 1:2,000 sublimate solution, dried, and then sprinkled with an ethereal solution of iodoform. Then it was covered with iodoform gauze, upon this sterilized mull crumpled together was placed and then the usual modified Lister dressing of our clinic. I still use sterilized gauze and cotton in fresh wounds instead of the moss-pillow.

The iodoform tampon, I believe, accomplishes three objects: First, complete control of hemorrhage, which seems important in preventing extravasation of blood into the fissures and infiltration of blood into the meshes of the arachnoid; second, it absorbs all fluid in an especially effective manner, and in this way it causes the approximation and agglutination of the subarachnoid spaces which have everywhere become opened; finally, I consider the pressure which the pieces of mull exercise upon the surface of the brain to be beneficial, because it prevents overfilling with blood, to which there is certainly the greatest tendency after the escape of the cerebro-spinal fluid. In cases in which the brain has become bruised very much, this procedure may also serve to prevent a further escape of brain substance and additional prolapse of the brain; such an indication would exist, for instance, in comminuted and complicated fractures.

On the second day after the operation, the dressing was changed and the iodoform gauze removed. Then the wound was sutured, an opening remained in the place of the former scar. The wound, with the exception of this opening just alluded to, healed by primary union. The filling with granulations and covering with a cicatrix lasted until the third week.

After the patient had recovered from the effects of the anæsthesia, and attempted to undertake certain movements on the evening of the day of operation, he was conscious of an interference with extension of the hand. This part of his upper extremity, which had previously always been in a condition of extreme extension, hung listlessly, and neither the fingers nor the hand could be extended, while movements of flexion had remained unimpaired. The centre which we had attempted to extirpate had really been found and removed in a sufficient and satisfactory manner.

But our operation did not have any influence upon the oc-

currence of the epileptic seizures. To be sure, there was an immediate cessation of the attacks, and the attendant and the patient's folks thought that they were weaker than formerly and did not last so long nor occur so frequently. But they were present and had not, as I had hoped would be the case, disappeared. The paralysis of the extensors of the fingers lasted about three months and then it slowly disappeared, so that in the fourth and fifth months, the patient could again extend the hand somewhat. We would have been glad to have secured an absence from epileptic seizures at the expense of a permanent paralysis of the extensors in question. I had counted upon an improvement and even a complete disappearance of the latter, basing this hope upon analogy in experiments upon animals; for the loss induced in animals used for experimental purposes is a transitory and temporary one, and tends to disappear the quicker, the smaller the portion of cortical area excised.

The case in which Horsley excised a definite psychomotor centre of the cortex of the brain is reported in his tables of April 23d, 1887. Unfortunately it is only described briefly in the manner of the following account; but it is worthy of notice on account of the manner in which the desired area in the cortex was located and found.

A boy of ten who was deficient mentally, suffered from a paresis of the left side of the tongue, face, and an interference with speech. Epileptic convulsion occurred from three to six times a day, commencing with twitchings in the left angle of the mouth. October 19th, 1886, trephining of the right parietal over the seat of the centre for the facial, which had previously been determined. Opening of the dura and determination of this centre through faradic stimulation. Then extirpation followed. Under strict Listerism and drainage the wound closed in the very shortest period. The drainage-tube was removed on the second day. The twitchings of the left angle of the mouth occurred during the second night after the operation and a general convulsion occurred during the third night after the excision. The seizures which occurred after this were less frequent and less severe than they had been; there were intervals of three days during which there were no paroxysms. Otherwise the general condition was pretty much the same.

Neither in my case, nor in that of Horsley just reported, was there any material improvement. In the latter case, altogether too short a period had elapsed after the operation, to be able to express an opinion concerning its success. But the first two cases, in which with the centre in the cortex the irritative epileptogenous zone was also removed, had been free from seizures for over a year when the author last referred to them.

The lack of success in the last two cases—my own and Horsley's—cannot be wondered at. The operation was undertaken in cases that were too old and too unclean. Horsley's was especially so, because an etiologicl factor which irritated the motor region—an injury to the head—was absent, and a tumor, which might certainly have been thought of, was not found. Since there was no full history of the case, the question as to how much the given symptoms warranted the diagnosis of an affection of the cortex, cannot be discussed here.

My own observation, also, is a real complicated one and differs in this respect from Horsley's first case, which was so classical. In the latter, the entire course corresponded exactly with that existing in the experiments upon animals: beginning in one centre, spreading to the next contiguous one, etc. In my case, it was different: the general convulsions followed upon the tetanic extension of the hand so rapidly, that a typical sequence could not be observed. Added to this there was the long duration in my case, and also the existence of secondary degeneration, rendered probable not only by the psychic condition of the patient, but also by the constant position of the right hand and the rigidity of the muscles of the right extremities during the intervals between the convulsions. Finally there were also absent manifestations which could be expected in the cortical forms of epilepsy described by Jackson: paralysis and pareses which are apt to follow the convulsions and even though of short duration, are still plainly developed. Finally no disturbances of sensibility, of taste, or of muscular sense could be demonstrated. It is not surprising, therefore, that my curative efforts did not succeed; but, at the same time, this fact should not serve to question the correctness of our propositions and deductions.

It seems Keen [97] was more fortunate recently:

The case occurred in a large and strong merchant, twenty years old. At the age of thirteen, he fell asleep in a chair in front of a stove and fell down, striking his head against the stove so forcibly, that, for a time, he lost consciousness and he had headaches the entire day. It cannot be ascertained which part of the head was injured at that time. Since the accident, he has had epileptic attacks which last a short time; the eyes would become staring, the head turned to one or the other side, and finally general very violent twitchings would occur. Keen believed he could feel a groove indicating a fissure in the right temporal region, when the head was shaved. The patient and his folks wishing it, he cut down upon this, but finding nothing but the canal of a blood-vessel, he did not proceed any further. Patient remained under his observation. In each of eight attacks, the convulsion began in the left arm and fingers. The thumb and fingers were separated, then flexion of the hand and elbow, then the convulsion extended to the left leg, then to the right, and finally to the face with marked deviation of the mouth to the left. Not until after this did the general convulsions ensue. In consequence of this constancy in the picture of the disease, Keen decided upon an extirpation of the centre for movements in the left hand. Operation May 30th, after determination of the position of the fissure of Rolando. After exposure of three convolutions and the fissures between them, an attempt was made to locate the position of the centre for the left hand by means of faradic stimulation—an attempt which succeeded admirably. Upon irritation of the most anterior of the convolutions, extension of the hand and abduction of the fingers ensued. The operator also demonstrated that the seat of movements at the elbow-joint and shoulder was above this centre, and that of mimicking movements was inferior to it. Keen excised the centre for the hand. Then the wound was closed and bandaged in the usual manner.

Immediately after the operation the patient was restless and had an epileptic attack during which the left hand was motionless and the left leg twitched first. Examination showed complete paralysis of the left hand, which continued for some time after. Sensibility of the hand and fingers had become changed, so that every touch was located correctly, but two points were felt instead of one only when the points of

the compass were separated for the entire length of a finger. June 3d and 4th, had attacks of the same character as he had had before the operation, though they were shorter in duration and less severe; another, June 7th. There was no movement of the left hand during the attack. June 28th, patient was discharged. July 20th, examination showed that the left hand was recovering its power of motion.

In summing up my exposition, I want to emphasize again the difference between those cases which represent pure pictures of traumatic cortical epilepsy and those in which characteristic attributes of the latter are wanting. In the former category, I place all those in which there is a constant commencement in one group of muscles, and in which this group is especially involved in the attack, in which there is a typical method of spreading upon the same and upon the opposite side of the body, and finally, in which there is temporary or prolonged hemiparesis and real paralysis in the group of muscles which is first involved in the epileptic seizure. To the other, the second category belong all other cases in which the attacks begin suddenly and lightning-like, the patient falling to the floor, bending the head backward, placing the trunk in a condition of opisthotonus, flexing the thumb and pressing the teeth tightly together; cases in which this condition of tonic spasm, after lasting only a few seconds, is followed by clonic spasms in which spasmodic distortions and twitchings occur simultaneously and violently in the face and in all the extremities.

We should be content to apply our surgical assistance only in cases of the first category. In saying this I do not wish to admit that, regarding the anatomical substratum of the disease, there is an essential difference between these cases and those of the second category in the sense that in one class of cases the cortex is the seat of the disease, and in the other it is the convulsive centre of Nothnagel. The circumstance that in every case the aura, whether visible, sensory or motor, occurs in spheres which are represented in the cortex of the brain, is undoubtedly a weighty point for the believers in the exclusive cortical origin of epilepsy. For us surgeons, another factor is the decisive one. As often as there occurs a good and entirely characteristic case of Jacksonian epilepsy

after injuries to the head, there are tangible changes in the cortex of the brain; and not only neuroses, but macroscopically changed diseased areas, recognizable to sight and touch, will be found. Our operative activity has a definite object: the removal of the diseased portion and with it that area of the cortex the irritation of which shows itself in the muscles which at first are the only ones affected. The accomplishment of this task is a matter for modern surgery of the brain. That it can be performed has been shown by three operations already done (Horsley, Keen, von Bergmann). Not until many cases have been operated upon, will it be possible to say whether this method of cure has acquired a permanent position in the achievements of our art. At present I can only state that it emanated from experiences in observations and experiments, and this, in the end, is the foundation for all our curative efforts.

I cannot speak in behalf of those surgeons who, depending upon the small amount of danger in our operative procedures, simply cut away and operate without thinking very much about it, in order to see and try whether it might not help. I shall always oppose such irregular proceedings. But I not only consider it permissible but proper to make use of, and apply at the bedside, the results of experiments upon animals. The cases of epilepsy caused by injury correspond to the manifestations of such experiments upon animals. If the extirpation of one of the cortical areas succeeds in animals, it must also be applicable in the analogous cases of human disease which are incurable in other ways. Since cortical epilepsy, *i.e.*, that form of epilepsy which has a similar course to that produced by electrical stimulation of a cortical area, if it occurs after injuries involving the roof of the skull, presents anatomical changes, it is the only form that is accessible to the knife of the surgeon. In wise limitation, I expect a real progress in brain surgery in these cases.

To traumatic epilepsy must be added a series of so-called epileptoid conditions. The author of the chapter on these conditions in the sanitary reports of Germany deserves great credit for having pointed out the frequency with which attacks of vertigo, for instance, occur after injuries to the head.

The epileptiform attacks of vertigo of which we are speaking are distinguished by the irregular intervals between their

occurrence, by trembling of the extremities, and by their association with palpitation of the heart and disturbances of digestion. Often they are followed by numbness of the head lasting anywhere from a minute to a day or by a persistent, severe headache. This form of vertigo was found in not less than 132 cases of grenade and gunshot wounds affecting the head, by the examining German military surgeons.

To this class belongs also the delirium occurring in attacks, which is known as the so-called "psycho-epileptic equivalent" of epilepsy, because it alternates with typical epileptic seizures. When these attacks develop after injuries to the head, especially after gunshot wounds we might think of operative treatment. But, from what has been said in the preceding pages, it is evident how little can be said about this endeavor. We have gained and opened up more prospects for the surgical therapeutics of epilepsy by advising an operation only in the cases produced by traumatism—cases which have a definite form, which commence with spasms in one and the same group of muscles and which are generally known as Jacksonian epilepsy; in the second place, the prospects for such surgical interference have been improved by our having set a distinct mechanical task: the extirpation of those cortical areas which act as a centre which, when irritated, causes spasms in the muscles first involved. The views and suppositions which underlie such a conception are only connected with the extirpation of the scar to this extent, that the latter guides the surgeon in the search for the field of operation, lying next to or over the latter and on this account having to be separated or removed.

In the cases of epileptic equivalents, there is even a greater absence of an indication of a localized starting-point of the spasm than in cases in which the seizures are general from the very beginning. There is, in these cases, no trace of an affection of a definite motor centre serving as the cause and origin of the disease; on this account the question of the removal of a cortical area exciting the attack, cannot be thought of. If this were done, there would only be one result: the substitution of one scar for another, the scar which we produce would take the place of that which had existed. I do not believe that we can associate a clear therapeutic idea with such an exchange. At the very best, without any real justifi-

cation, we might substitute for the old scar, which we believe to be an irregular one and one which irritates the cortex, a new one that is smoother, cleaner, and less irritating and pressing. Now I do not wish to deny that scars may develop, especially in the cases of gunshot wounds of the head alluded to above, and that we may attribute to these an irritating and disturbing influence upon the brain; such, for instance, may be firm, callous masses of connective tissue in which bony fragments which have become loosened and pressed into the brain, have become impacted, or cystoid degenerations of cicatricial tissue sunk deeply into the cortex of the brain. But there is only one way of recognizing, or at least of supposing such unfavorable conditions to exist: the area symptom. If symptoms of irritation or exclusion referable to a distinct cortical area are missing, the justification for our attack is wanting. On this account it is that the supposed scar in the disease now being considered was not always found, and that often no change in the brain and its membranes, or even on the bone, was discovered. I will content myself with referring, in this connection, to the patient trephined by me who died afterward, and an account of the autopsy upon whom is given on a preceding page.

CHAPTER V.

THE PUNCTURE OF THE VENTRICLES OF THE BRAIN AND THE OPERATIONS FOR THE RELIEF OF BRAIN PRESSURE.

I AM aware that the preceding pages have not exhausted the entire province of the operative treatment of diseases of the brain. A series of pathological products make their appearance and collect upon the surface of the brain. If these masses are situated in the region of the central convolutions, they may produce area symptoms which enable us to be certain of the location of the disease; but the simple localization of the disease does not acquaint us with its nature. The latter is only determined when we know whether the case is one of tissue degeneration in the involved cortical areas, or an exudation or a new growth which possibly may be removed and is the cause of disease and disturbance of the function of this region. If the diagnosis assumes the existence of removable products at the operation, we would still have to weigh all that which has been discussed in connection with the extirpation of masses of tubercles and of gummata. Ceci [98] called attention to operative interference in cases of hemorrhage coming from a pachymeningitis hemorrhagica chronica, and presented such a case before the meeting of Italian surgeons. As is well known, Huguenin has again endeavored to show that the pachymeningitic changes in cases of dementia paralytica are due to primary subdural hemorrhage. If his theory be correct, we must admit that the traumatic rupture of the vessels passing through the subdural space may have the same effect. As far as I know, Birch-Hirschfeld, in his text-book on pathological anatomy, was the first to describe a case of pachymeningitis hemorrhagica occurring beneath the seat of a cured fracture of the skull. The case was the post-mortem examination of a child six months old, who had received a fracture at birth. Occasionally we meet with observations of cases in

which changes in the dura, corresponding to typical and pathological pachymeningitis hemorrhagica, occurred—as, for instance, the case of Silvestrini's, reported in the Paris Academy of Medicine;[99] in this case, a boy who died fifteen months after receiving a depressed fracture, the dura was found thickened, opaque and soft, and beneath it an old, almost completely organized blood-clot in the left temporal region, beneath the seat of the fracture. Ceci's case occurred in a farmer, fifty-two years old, who fell and received a severe injury to the right parietal bone, which healed without any treatment. Two months later, a rapidly progressing paralysis of the left side developed, commencing with a weakness in the left foot; at the time Ceci was consulted, the paralysis was complete. The tongue and uvula were drawn toward the left side; there was incontinence of urine; patient lay in a semi-comatose condition. The diagnosis of formation of a cerebral abscess in the motor region seemed most plausible and was supported by the existence of a cicatrix and shallow depression over the right parietal bone. After the trephine had been used, the changed appearance of the dura, which was firmly adherent to the bone, became manifest. When incised, it appeared very much thickened and of a dark blue color. Beneath it was fluid blood mixed with old coagula. Puncture in all directions with a hollow needle failed to reveal the presence of pus. The patient recovered from the operation; his paralysis disappeared entirely, and six or eight months after the operation, when he was presented at the meeting of Italian surgeons, he had only a slight paresis of the left hand.

Among Macewen's cases, there is one which is somewhat similar to that just described. Hoffmann[100] also reports two cases of neuropathic disturbances following injuries to the skull, in which the area symptoms did not occur immediately, but days and weeks after the injury. But I cannot consider cases of this sort under the same heading as those that we have just been describing, for they deal with direct results of the traumatism appearing during the progress of recovery from the injury itself. Their consideration belongs to the province of head injuries.

It is different in cases of fixed, exactly localized headaches. Here the connection with the traumatism is not so easily made out, and in some cases it is impossible to trace this. As

is well known, this variety of headache, on account of its severity and obstinacy, has led the older surgeons to make use of the trephine. Most of the cases which are enumerated under this heading, probably have no connection with the brain. Contusions and bruises of the skull often leave behind headaches; these sometimes trouble the patient continuously, sometimes only at times like a neuralgia; they disappear after a shorter or longer period, sometimes upon covering the painful spot with an ice-bag and upon giving a hypodermic injection of morphine during the attack.

Three years ago, I treated an especially stubborn case of this sort. The surprising character of what was found on the skull of a girl, then twelve years old, warrants my alluding to the case here: Six months previously, a stone had fallen upon the right side of the skull without having caused any abrasion of the skin. At the point of contact a swelling of about the size of a walnut developed, which soon disappeared. Some weeks after, pains began which were located at the point at which the swelling had been. A variety of remedies having proved of no avail, the suffering patient was brought to me. After applying an ice-bag continuously day and night and causing a diminution but no disappearance in the pains, I made an incision down to the bone over the painful spot and separated the periosteum with an elevator. The surface of the skull was of a dark blue color, resembling the appearance of blood-clot showing through a thin lamella of bone. As a matter of fact, the bone in this situation for an area of about two centimetres had become reduced to the thickness of letter paper, so that I could break it away with the point of the scalpel. A quantity of tarlike, dark blood escaped, after which the dura, pulsating lively, came into view. Hemorrhage was controlled by pressure of a wad of gauze. I sewed up the wound and dressed it in the usual manner. Upon recovering from the effects of the chloroform, the pain had disappeared and it has not since returned. What changes had taken place in the bone, and why it was thinned out so much by an extravasation of blood into the diploë or above the dura, I do not know. I was reminded of this case by one of Horsley's, which has recently been published; it is the seventh case in his table: A man of thirty-seven suffered from fearful headaches for three years; the pain was so severe as to incapacitate

him from any kind of work. It was limited to one spot in the parietal region. November 12th, a button of bone was removed from this situation. This piece of bone was very thin and perforated, no doubt absorbed by a Pacchionian granulation. There were no pains after this.

Cases like the ones just described certainly belong to the surgical curiosities; on the other hand, the question of operative interference in cases of increased brain pressure is important as applying to an extensive series of diseases of the brain and is decisive as regards their therapy. It may be considered as belonging to two sets of cases of endocranial disturbances: first, tumors of the brain, and second, acute increase of the liquor cerebro-spinalis—the rapidly developing distention of the ventricles with fluid as a result of tubercular basilar meningitis.

The influence of the removal, *i.e.*, the diminution of endocranial pressure upon the course of tumors growing in the interior of the skull, and the immediate effects of such a diminution have been beautifully described by Jastrowitz [101] last year. Among a large number of cases of tumors of the brain, death is not produced by the destruction of parts of the central nervous system important to life, but because the rapidly growing tumor constantly occupies more and more space in the skull and thus leads to disturbances of circulation due to so-called brain pressure. This increases to such an extent that the most important functions of the brain are interfered with and lost, and thus the death of the patient results. We see the enormously rapid effect which the relief of pressure in the cavity of the brain has upon a patient who is already near death, in cases of injuries to the head; it is seen, for example, in the removal of blood which has escaped from the torn middle meningeal artery and which has collected between the dura and the bone. A very striking result ensues. The patient is in a deep sleep, with stertorous, labored, and occasionally intermittent breathing, and a pulse of 30 to 40 per minute. We open the skull, not so much to check the hemorrhage as to remove the blood; we find clots of the size of the fist and fluid blood between the bone and the dura which has become widely separated from it; we remove this extravasation—and at once our patient awakes in full, clear consciousness. his pulse increases in frequency to 90 or more, and his respirations become

free and regular. These are the results of the most charitable operation imaginable. The existing relations are the same as in experiments. The more wax we force into the cranial cavity of an animal experimented upon, the higher will the blood-pressure rise and the slower will the pulse become, until the stage of irritation of the vaso-motor and of the vagus centres passes into that of paralysis, and a pulse of increased rapidity takes the place of the slow one. Even in the beginning of the latter stage, we can cause a disappearance of all dangerous manifestations and can keep the animal alive, by removing the wax. This is the fundamental experiment from which we proceed and upon which we base our hopes of diminishing operatively the increased brain pressure occurring in any disease.

In the experiment, we place the cause of the diminution of space between the bone and the dura in order to be able to remove it easily if the object of the experiment should demand this. In the same manner, those pathological collections and formations which are situated between the dura and the bone, as, for example, an extravasation from the middle meningeal artery, are not accompanied by any difficulty in removal through an opening in the skull. If this has occasioned manifestations of brain pressure, its operative removal must relieve this pressure, just as was the case in the experiment. This really occurs in the formation of gradual increase of hemorrhage from the middle meningeal artery and also in removal through the operation. In the rare cases in which pus collects between dura and bone, the result is the same or at least similar. I have already referred to such a case above. The patient had headaches, vomiting, and slowing of the pulse; all these disturbances disappeared upon the formation of an abscess beneath the soft parts of the skull. In opening the latter, a perforating tubercular osteitis was found; hence it is probable that the manifestations of brain pressure disappeared as soon as the pus, which was originally endocranial, had formed and had taken an outlet through the opening in the diseased bone. The tumors which grow from the dura must behave in an analogous manner. As a matter of fact, cases have been known, in which headaches, vertigo, and vomiting became less marked or even disappeared entirely, as soon as the tumor had perforated the skull and had become visible and tangible.

It would seem plausible that, in analogous cases in which the size or infiltration of the tumor renders its removal impossible, the threatening brain symptoms would cease upon removal of the portion of the skull lying immediately above the growth. I should imagine, however, that this procedure could be of little value in cases in which the sarcoma is situated at the base of the cranium. In the treatment of these cases, we must not forget that the brain with its annexes, its blood-vessels and their contents, and finally its liquor cerebro-spinalis, is not only inclosed in the rigid-walled cranium, but that it is also encapsulated by the dura mater; the latter, even though it is less firm and unyielding than the bone, can be stretched and dilated only slightly. Consequently exposure of the dura would be of little value in the removal of increased subdural tension. Even the opening of its capsule does not act any too much or any too long, in the sense of effecting a diminution of pressure; for the portion of the brain which is forced into this opening soon applies itself to the margins of the defect and thus a dense and firm barrier again results. In the observation of Heath, described more fully above, in which an operation had to be stopped and abandoned because a tumor was found adherent to the bones of the base of the skull, the headaches, epileptic seizures, and paralyses which had previously existed, ceased in consequence of the extensive opening of the cavity of the cranium. However, in this case, the opening in the frontal bone permitted the unembarrassed growth of the tumor emanating from the anterior fossa out of the cranial cavity. I doubt very much whether an opening with the trephine would ever diminish brain pressure in cases of subdural tumors and extravasations of blood, as, for instance the hemorrhages into the substance of the brain caused by disease of the cerebral arteries.

On the other hand, we may look for a different effect in the operative influence upon excessive brain pressure under a certain other condition: relief, through removal of excessive, *i.e.*, pathologically formed or collected, liquor cerebro-spinalis. Such a condition is known to exist in the acute hydrocephalus of tubercular meningitis. It is not the small, miliary nodules along the blood-vessels which occasion the danger, but it is the inflammation of the plexus and its product—the fluid, which is rapidly poured into the cavities of the brain. In other words.

the most prominent and important phase in the picture of this disease is the rapid effusion of fluid and not the development of tubercles. This effusion, more than anything else, is the cause of danger. With increased effusion, *i.e.*, increase in endocranial pressure, the irritation symptoms of the beginning pass into the paralytic symptoms and the child then dies from the latter. In these cases, we can actually see how the increase in the effusion into the ventricles weakens, slows, and even stops the circulation. When the effusion increases, the fontanelle of the child bulges and pulsates plainly, as soon as the pressure from within begins to manifest itself; but while the heart still acts vigorously and the power of this organ is complete and normal, the pulsations in the fontanelles become weaker. The intracranial pressure has become so great through the increase of the ventricular cerebro-spinal fluid that it offers an impediment and finally entirely obstructs the access and course of the stream of blood.

This would certainly suggest the idea of relieving and ameliorating the course of the disease.

There is also an additional consideration: Like that which collects in the cavities of the brain in miliary tuberculosis of the pia mater, there collects an extensive exudation in the cavity of the peritoneum in cases of miliary tuberculosis of this serous membrane; the ascites of tubercular peritonitis forming slowly at times, rapidly at others. It is well known that puncture and incision in these cases have opened up prospects of successful treatment which *a priori* would have been considered impossible. These achievements have anticipated theory; we really know nothing regarding the manner in which the rapid and thorough emptying of the peritoneal cavity favors the retrogression of tubercles. Even though we attribute the favorable course of the disease to the general improvement in circulatory relations produced by the removal of pressure, we have not explained the influence of this upon the fundamental process. It is certain that the circulatory relations of the interior of the skull and also of the diseased pia mater are materially improved after extensive withdrawal of cerebro-spinal fluid from an effusion into the ventricles.

Will this improvement also cause a retrogression or at least a standstill in tuberculous processes? The attempt to influence the latter, in this sense, by puncture or even by incision

into the ventricles, based as it would be upon the successful analogy in tuberculous peritonitis, could not *a limine* be discountenanced. On this account, in the following case to which I was invited by my colleagues Drs. Gerhardt and Haussmann, I did not offer any objection to combating the paralytic stage of a tubercular meningitis, accompanied by an extensive effusion into the ventricles, by a large puncture of one of the lateral ventricles. I am indebted to the kindness of the family physician, Dr. Haussmann, for a history of the case from which the following notes are taken:

The patient was a girl three years old, who had always been delicate; she did not commence to walk until the end of the second year; she suffered repeatedly from intestinal catarrh; she was backward in her general development. In March, 1887, she had a severe attack of gastro-enteritis; in May, 1888, she had chicken-pox. Then, after an error in diet it was said, there occurred an attack of clonic spasms lasting two hours; this was repeated on the following days, though with diminished intensity. Most often she vomited the nourishment which was given her.

July 4th, the diagnosis of tubercular meningitis was made: the child complained of headache, was constipated, weak, and sleepy. This condition became worse from day to day. July 12th, an irregularity in breathing was first noticed; July 13th, the pulse fell from 120–130 down to 84–92. The pulse, which had been regular, now intermitted frequently. When I saw the child, July 15th, it lay in a deep sopor, alternating with restlessness and groaning. Pulse 72, intermittent; respirations of an exact Cheyne-Stokes type with very long pauses. Twitchings in the left arm which is flexed at the elbow; also in the flexed fingers of the left hand. Eyes closed. Pupils dilated and do not react.

As the doctors in charge, reasoning as I have done above, decided to interfere operatively, I began trephining at once. I removed a piece of bone from the skull just above and a little the inner side of the frontal eminence. I then inserted a long hollow needle into the substance of the brain from before backward and slightly downward and inward, until the cerebro-spinal fluid escaped in a stream. I allowed all to escape that would, before I removed the needle. The opening in the

skull was kept open by a tampon of iodoform gauze. The cerebro-spinal fluid which had escaped measured 20 c.cm. and was perfectly clear.

Immediately after the operation, after being placed into a warmed bed, the child's respirations numbered 40 and were regular; pulse 104, regular and not intermittent. Immediately after there were slight twitchings in the right arm; in the course of the day the frequency of respirations increased to 60, that of the pulse to 128; in the evening, pulse 84, temperature 101.4°.

The pieces of sterilized mull which were placed over the iodoform gauze wad were saturated so thoroughly with the constantly escaping cerebro-spinal fluid that, at the change of dressing in the evening, they felt like a wet sponge. On this account, the dressing is changed several times a day.

During the night after the operation, the little patient took some milk for the first time in days, and did not vomit. She grasps the bottle and brings it to her mouth. She also takes some finely-chopped meat. For days after, there was no vomiting, while before the operation this was exceedingly frequent.

She opens her eyes, which before she had kept closed. Pupils are less dilated and they react to light. The child sits up in bed, though drowsily. The improvement continues during the second day after the operation, the cerebro-spinal fluid still trickling through the small punctured wound. Evening of the 13th, temperature 101.4°, pulse 128; the child took some milk and bouillon and some meat. On the third day, the breathing again irregular and intermittent. Feet are cold; pulse 112 to 116.

July 19th, drowsiness more marked; after twelve respirations there is a pause. Pulse again intermittent. No reaction upon pricking the finger and arm with a needle. Less discharge of cerebro-spinal fluid. Frequent sighing. Grinding of teeth. On the 20th, early, twitchings in face and both arms. Pulse 100. Respiration intermittent. Since nine in the morning, there have been tonic spasms of the neck, masseter, hands and feet. At twelve, temperature 104.4°. Muscles become less hard and tense after inhalations of chloroform. Soon, however, the spasms reappear: at three in the afternoon, the child died. No autopsy was allowed.

We were not successful in curing the little patient. But the improvement after the operation was unmistakable and striking. Breathing became regular, the pulse increased in frequency, the vomiting ceased, and there was again a desire for food. Furthermore, the operation showed that the dilated lateral ventricle could be reached with certainty by means of the course selected. Perhaps it would have been better to have made use of a thicker instrument than the hollow needle that was employed, and to insert a drainage-tube into the canal formed in this manner. But even without this, the fluid did not cease to flow off and seemed to escape freely. My surgical invasion undoubtedly accomplished its task—the diminution of the dangerously increased intracranial pressure. I do not deny that the constant escape of liquor furnishes an open channel for impurities of an injurious nature which exist in atmospheric dust and which are attracted by the wet dressing into the cavities of the brain. The dryness of the dressings, which is one of the most important aids to antisepsis, is absent here; hence the protection of the wound is accompanied by greater difficulties than is usually the case. On this account, such protection was not accomplished in the case operated upon in my clinic and which is described above. However, with more attention and care, we have been able to combat this danger in other cases, as, for instance, in two cases in my practice, in both of which the lateral ventricle had undoubtedly been opened into and in which for days great quantities of cerebro-spinal fluid soaked the bandages. Horsley has accomplished still more in this direction in his case of tumor of the spinal cord which he operated upon recently, in which he had to combat all the difficulties of this escape of fluid.

Not every case of tubercular peritonitis has been cured by laparatomy. Every explanation of any peculiarities in my emptying of the lateral ventricle of the brain and of the membranes of the brain in this case, as well as the point from which the meningitis started and how far it had progressed at the time of the operation, is wanting, owing to the fact that no autopsy could be held.

Just as in the provinces of abscesses and tumors of the brain, of encephaloceles and of epilepsy, there must be great limitation if brain surgery is to occupy a prominent position

in mechanical therapeutics, the operative diminution of brain pressure will be applicable and of benefit to the patient in but a few selected cases; and when thus employed, it will be able to secure the position among modern operations to which it is entitled. If the motto of our surgery of the brain be "much criticism and great care," further work in this direction will be and will remain successful and blessed.

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